SURGICAL TECHNIQUE

and Principles of Operative Surgery

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Foreword

THE RAPID STRIDES which have characterized the amazing progress of surgery during the past half century began with the great impetus given it by the epochal contribution of Lord Lister, in 1867, when he propounded the principles of antisepsis. The high toll of infection during the Pre-Listerian Era precluded the possibility of elective surgery and most operations performed were crude and hazardous emergency procedures. However, the establishment of the fundamentals of asepsis so widened the scope of surgery that for a period of a quarter of a century surgeons throughout the world began the development of technical procedures based on anatomical principles. The importance of "cellular pathology," which had been introduced recently by Virchow, was readily appreciated, and the recognition of disease processes in the living body became essential.

Thus, during this early period great emphasis was placed upon the importance of anatomy, pathology, and manual dexterity. Consequently a surgeon's training consisted primarily of studies in the anatomical laboratory and in the postmortem room. A basic knowledge of anatomy, a fundamental understanding of pathologic changes, and the perfection of operative technique remain yet undeniable desiderata for the adequate and proper performance of the surgeon's function.

More recently, however, the importance of physiology has been realized and the rapid advancements that have marked surgical progress during the past few decades have been due largely to the continuous endeavor by physiologically minded surgeons to correlate experimental physiologic alterations with clinical manifestations. The constantly increasing number of presumably non-surgical diseases which recently have entered the ever-widening realms of surgical endeavor clearly illustrates this modern trend. Obviously, a fundamental knowledge of normal physiology is essential in the rational and comprehensive correlation of disordered physiologic functions consequent to pathologic changes. Upon this is founded rationale or the logical basis of a procedure which forms the essence of present-day surgical therapy. Thus, the modern surgeon can no longer be content with an adequate knowledge of anatomy and pathology; to these basic sciences he must add a thorough understanding of physiology.

The complete removal of a disease process and the proper and expedient performance of an operative procedure are not the only concerns of the modern surgeon. Of equal importance is the careful and prescient preparation of the patient. He is as interested in making the patient safe for the operation as the earlier surgeon was in making the operation safe for the patient. Moreover, the incidence of operability has increased enormously by such scrupulous pre-operative preparation. Appropriate operative procedures for previously considered inoperable conditions can

now be performed successfully by the institution of these anticipatory measures. Of no less moment is postoperative care. Unfortunately, the false conception that nausea, gas pains, and distention are the inevitable consequences of abdominal section still prevails in many localities. However, the rational clinical application of therapeutic measures based upon physiologic principles obtained through experimental investigations has completely eliminated such postoperative discomfort.

It is gratifying to have a book on operative surgery in which not only the technical phase of the operative procedure is lucidly described and admirably illustrated, but in which the rational indications for the operation based upon the physiologic principles involved in the disease are thoroughly considered. Too many texts on operative surgery have consisted primarily of mere descriptions of operative technique with little or no consideration of the underlying pathologic-physiology, the logical basis for the procedure, or the adequate preoperative and postoperative therapy. Thus, the successful removal of a common duct stone in obstructing jaundice depends not only upon dextrous operative manipulation but also upon the proper preoperative preparation of the patient and the institution of careful postoperative care.

The significance of Atraumatic technique, careful hemostasis, and gentle handling of tissues, which are essential in any operative procedure, are particularly emphasized in Dr. Partipilo's "Surgical Technique and Principles of Operative Surgery." Also, the significant physiologic alterations of disease processes so important in the rational consideration of surgical therapeusis are thoroughly discussed.

The indications for an operative procedure in different conditions vary considerably, depending largely upon a thorough knowledge of pathologic changes and the consequent physiologic derangements. An entire chapter is devoted to a consideration of the disturbed physiology in intestinal obstruction. Another chapter is concerned solely with the considerations of principles of gastric surgery. Surgery of peptic ulcer may seem orthodox to many surgeons. However, those of wide experience with operative results in peptic ulcer are firmly convinced that individualization in the treatment of this disease is of paramount importance. Throughout the book special emphasis is placed upon the rationale and basis for the indications of particular operative procedures in individual cases.

Because of his intensive anatomical and sugical training, Dr. Partipilo is exceptionally well equipped to present "Surgical Technique and Principles of Operative Surgery." For three years he was an associate in the Department of Anatomy of the Loyola University Medical School and for twenty-six subsequent years, Clinical Professor and Director of the Division of Operative Surgery and Surgical Anatomy in the same institution, and Director of the Chicago Post-Graduate School of Surgery. His practical presentation of the subject in such a lucid and logical manner is the result of his extensive teaching and clinical experience. Dr. Partipilo's collaborators are also practicing surgeons of wide experience, actively engaged in

The previous editions of Partipilo's "Surgical Technique and Principles of Operative Surgery" have been real contributions. The present edition is an entirely new book and is up-to-date in every respect. Important features of the book are the complete bibliographies which follow each chapter and a series of questions which serve to impress upon the student the important phases of the subject. Both of these features are extremely valuable for the undergraduate as well as for the graduate student.

Dr. Partipilo has been extremely successful in his choice of collaborators and yet the work has a uniformity which shows the effect of a good author. The work is clearly and concisely written, making it very easy to read.

The sixth edition of Dr. Partipilo's monograph is a contribution to medical literature. Because of the simplicity and clarity with which it is written and the profuse illustrations, it will be of great value to the undergraduate medical student and to the practioner of medicine.

ALTON OCHSNER, M.D.

cedures are freely suggested. Both the author and his fellow-authors have confined the descriptions of technique to their own surgical methods and practice. Furthermore our objective has been not only to tell how a procedure is done, but we have attempted to explain the reason for each step or maneuver.

The illustrations, 1235 in number, have been done by Mr. W. C. Shepard, Mr. Hooker Goodwin, Miss Virginia Samter and Miss Romona Morgan. The painstaking efforts by the artists in combining anatomical and technical details with scientific accuracy add greatly to the value of the book.

In this Edition, thirty-eight new chapters have been added. Three of these are by Kenneth Weiss on the general principles of anesthesiology with which the surgeon should be familiar. Dr. Joseph P. Concannon has contributed a chapter on radioisotopes and radiation therapy in surgery; another on the use of radioactive iodine in cancer of the thyroid gland. In collaboration with Dr. Gerald Nora he has contributed a chapter on the physical basis of radioactive isotopes, and another with the (senior) author on the subject of tumors of the thyroid gland. The increasing emphasis, in recent times, on the use of isotopes and radiation therapy in the diagnosis and treatment or as adjuvants to the surgical management of diseases seems a justifiable inclusion of this work in this Edition.

The advancements made in the field of cardiac surgery and surgery of the great vessels have been the outstanding surgical accomplishments of the past decade. This factor is reflected in this Edition by the contribution of nine chapters on these subjects by Dr. John L. Keeley. These include the following: constrictive pericarditis; patent ductus arteriosus, coarctation of the aorta, vascular compression of the trachea and esophagus, mitral stenosis and regurgitation, pure pulmonic stenosis, aortic stenosis and regurgitation, tetralogy of Fallot and auricular and ventricular septal defects.

As in the field of cardiac surgery, thoracic surgery has also made tremendous improvements during the past ten years. In this Edition the entire work on surgery of the chest has been completely rewritten by Dr. George W. Holmes. He has contributed nine chapters on the following subjects: physiological principles; surgical anatomy; diagnostic procedure; preoperative care and evaluation of the patient; thoracic incisions; technique of pneumonectomy (lobectomy, segmental lobectomy, wedge resection, decortication of the lung, thoracoplasty and chest wall resection); surgery of the diaphragm; surgery of the thoracic esophagus and postoperative care and treatment of postoperative complications.

The chapter on surgery of the breast has been completely rewritten by Doctors Louis River and Joseph Silverstein. Dr. Steven O. Schwartz has contributed a chapter on the anatomy, physiology, and disease of the spleen. This includes a discussion of the indications for splenectomy. Dr. Robert Schmitz has contributed a description of the technique for splenectomy and a chapter on the management

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SURGICAL TECHNIQUE AND PRINCIPLES OF OPERATIVE SURGERY

THE PAST AND THE FUTURE OF SURGERY

The art and science of surgery is progressive and constantly changing. Its history may be divided into four periods: (1) the philosophical period; (2) anatomical period; (3) the pathological period, and (4) physiological period.

1. The philosophical period began with the dawn of history and ended with the Renaissance. During this period Medicine was under the influence of Hippocrates (460 B.C.) and Galen (130 A.D.), and surgery was of no consequence; a surgeon was no better than an outcast.

2. The anatomical period began with Leonardo da Vinci (1452–1519) the great Italian painter, sculptor, architect, engineer, philosopher, and the first to make anatomical drawings. Except for Da Vinci the 15th centry was entirely devoid of interest to the surgeon. The 16th century produced Andreas Vesalius, (1514–1564) the father of modern anatomy, Gabriel Fallopius, Jacobus Sylvius, Bartolommeo Eustachi, Fabricius, and Ambroise Paré, the father of modern surgery.

The 17th century was the golden age of anatomical research. During this period individual investigation was at its height and many epochal making discoveries were made. It is also noted for the long list of illustrious names that left an indelible imprint in the annals of medical history. Of these, William Harvey (1578–1657) stands out as a towering giant among a galaxy of great men. The announcement of his discovery of the circulation in 1628 was the most momentous and provocative event in the history of medicine. His classical work, "The Movement of the Heart and the Blood," did more than just describe the circulation. It shattered old theories and initiated the spirit of investigation. It may be said that Harvey was the precursor of modern physiology. The microscope was also invented during this period, hence we find microscopic anatomists such as Marcello Malpighi (1628–1694), the founder of histology, Van Leeuwenhoek, Swammerdam, Hooke, Kircher, and others who made historical contributions. To refresh your anatomical memory, here are a few more eminent names: Stensen, Peyer, Brunner, Willis, Wirsung, Wharton, Glisson, Cowper, Pacchioni and De Graaf. I would pity the poor student of anatomy who does not know of the contributions made by each of these! This was indeed the age when individualism was at its height and every subject was investigated and scrutinized. Our anatomical friends were fit companions for their contemporaries—Shakespeare, Bacon, Milton and Galileo.

Although surgery was more substantial, nevertheless the actual practice of surgery was at a low ebb, and surgeons were not yet considered qualified to enter the society of the physician. Surgery had to mark time, waiting for the full development of pathology, physiology, bacteriology and anesthesia.

3. Pathological period: The 18th and 19th centuries placed surgery on a scientific basis. Pathological anatomy as a distinct branch was originated by Giovanni Battista Morgagni (1682–1771). His most important work, "The Seats and Causes of Disease Revealed by Dissection" was published in 1761 when Morgagni was 79 years old. It consists of five books of letters, 70 in number, describing the morbid conditions of six hundred and forty dissections. For the first time postmortem findings were concisely correlated with clinical records, the true foundation of pathological anatomy. A worthy successor to Morgagni was Matthew Baillie (1761–1823), whose publication, "Morbid Anatomy," was the first of its kind published in the English language. Except for the contributions of these two pioneers, pathology as a science did not flourish until about the middle of

surgeons not only applying physiological principles to their work, but also carrying out experimental work in the laboratory. The pioneer surgeons of this school were: Theodor Kocher, William Stewart Halsted, George W. Crile, Anton von Eiselsberg, Harvey Cushing, Rudolph Matas, A. J. Ochsner, John B. Murphy, Charles and William Mayo and Sir Berkeley Moynihan. They were the prime movers of the conception that the operation must not only be made safe for the patient, but the patient must be made safe for the operation. The fruition of this inital effort has been made possible by the clinical application of the extraordinary iadvance ments made in all the basic sciences during the past few decades.

PHYSIOLOGICAL TREND

Thus surgery has passed through various stages and has developed concomitantly with advancements made in anatomy, pathology, physiology and correlated sciences. Today, as a result of the enormous contributions to the study of living tissue and its bearing on the living organism, the surgeon is directing his attention to morbid physiology in his endeavor to solve his clinical problems and to make the patient safe for the operation. Many baffling clinical problems of the past have been solved and are at present treated surgically. In this group are included diseases of the neurovascular system, the nervous system, the pancreas, liver, heart and in certain conditions of the reticulo-endothelial system in which the spleen assumes the chief role. Appreciation of physiology of respiratory mechanism has made surgery of the organs within the thoracic cage as safe as abdominal surgery. The increased knowledge relative to body metabolism, vitamins, biochemistry, antibiotics, and nutritional requirements of the patient, has tremendously widened the applicability of surgery. Many conditions which were considered hopeless a few years ago are being treated surgically as a matter of routine; this includes patients with heart disease, the senile, and patients with jaundice. When considering surgery in the advanced age-group the surgeon should consider the physiological status rather than the patient's age. Many young adults are physiologically older than their actual age therefore greater operative risks. The application of surgery to children and infants has also been widened. One should bear in mind that the physiological status of an infant is entirely different to that of an adult. Therefore, it would be extremely hazardous to treat an infant in the same manner as an adult. As Sir Lancelot Barrington-Ward put it, "The adult may be safely treated as a child, but the converse can lead to disaster."

Undoubtedly the patient is being made safe for surgery; however it should be stressed that while making the patient safe for the operation the surgeon should be careful not to neglect the original complaint. Too aggressive and prolonged preoperative preparation may become another form of procrastination which may eventually cost the patient's life. For instance, prolonged intestinal suction for the treatment of intestinal obstruction may prove fatal because the surgeon fails to recognize that the cause of the obstruction is due to a gangrenous loop of bowel.

It may seem trite to reiterate that the object of a surgical operation is either to save life, to correct or restore the function of an organ, or to relieve the patient of disabling symptoms. The surgeon must have these objectives in mind while operating if he hopes to attain the most satisfactory results. In an "emergency case" operative interference is usually indicated in order to save life, therefore, do no more than is necessary to save life. Unless the exigencies of the case demands

ANATOMICAL TREND

It seems hardly necessary to stress the importance of a thorough knowledge of anatomy. Nevertheless, there are many surgeons who, because of inertia or self-sufficiency, are found wanting in knowledge of this indispensable basic science. In pre-anesthetic days anatomy was of paramount importance, and all great surgeons were invariably great anatomists. Operations then were performed under the most adverse circumstances. The patient was bound and held down by the attendants while the surgeon, although tense, operated boldly, deftly, and with tremendous speed. In the meantime the terror-stricken patient was screaming and shrieking, and in the ultimate throes of pain he would faint just as the operation was completed. A knowledge of anatomy was not only a necessity, but of momentous importance to the surgeon, and his reputation was judged by the number of minutes in which he could amputate a limb.

With the advent of anesthesia there was less need for speed, and at the present time a surgeon operates calmly and with deliberation, knowing that his patient is in a state of blissful sleep. However, to operate with calmness and poise a thorough knowledge of anatomy is just as essential now as in pre-anesthetic days. There are many who, while admitting the importance of anatomy, have not performed any systematic dissections upon a cadaver since their freshman year in medical school. Their knowledge of anatomy has been derived from observations while operating upon the living body. At best, information thus gained is not only inadequate

and incomplete, but also dangerous. A surgeon with such an impoverished knowledge lacks one of the fundamentals required of a good operator.

PATHOLOGICAL TREND

In the pre-anesthetic days pathology consisted of postmortem studies, whereas in the wake of anesthesia and aseptic surgery, "living pathology" became a motivating force. As more and more regions of the body were being explored new operative procedures were devised and at the same time the surgeon became better acquainted with "living pathology." As a consequence great surgeons became great pathologists and the gateway to a surgical career was through the portals of the pathological laboratory. Until recent times, due to lack of experienced microscopists, microscopic diagnosis was not relied upon and surgeons made their own pathological interpretations based upon the gross appearance of the tissue. However, this could not prevail for science had to pass from the descriptive to the experimental stage and pathology from the gross to the microscopic.

Today every hospital regardless of its size has its qualified pathologist who renders accurate pathological diagnosis. Because of this the surgeon has developed an apathetic attitude toward pathology and depends upon the pathologist for a final diagnosis. As Bloodgood stated in 1931, "We are approaching the period when this great pathological knowledge and keen ability to distinguish the benign from the malignant at the exploratory operation is of fading value. . . . If this generation can become just as proficient in the microscopic differentiation of diseases exposed in the operating room by the knife, as Halsted and the great surgical pathologists beginning with Velpeau were expert in the recognition of benign and malignant conditions by their naked-eye appearance, there will be very few mistakes of incomplete operations for malignancy when the lesion is operable or complete operations for non-malignant growths." Pathology is one of the corner-stones of surgery and every surgeon should devote a considerable portion of his time in the pathological laboratory studying with the pathologist not only the gross appearance of the specimen removed at the operation but also its microscopic appearance. In this manner he will develop a more solid foundation for the exercise of his surgical judgment while operating.

MANUAL DEXTERITY

The ideal surgeon must not only be a good operator but also a master technician. He must be on the alert and be ready to meet unexpected conditions, make quick decisions, and finally execute an operative procedure with poise and calmness. To operate with calmness the surgeon must have an implicit faith in his ability to carry out any procedure indicated, regardless of the difficulties encountered. There are surgeons possessing many good qualities who operate clumsily and awkwardly because they have not developed manual dexterity. At times this may be due to lack of natural aptitude; more often it is due to insufficient training and unwillingness to devote time toward developing coördinated movements and efficiency. The movements of a dexterous surgeon are agile, purposeful, rhythmical, and apparently executed without effort or exertion. Manual dexterity is attained when the movements of the hand passes into the realm of the subconscious. Such proficiency can be achieved by long and incessant practice. The surgeon owes it to his patient, to himself, and to his profession to devote part of his daily activities

the 10th or 14th day. (3) The final phase is the formation of a firm adult fibrous tissue or scar.

The rate and extent of the length of the "lag period" and phase of fibroplasia varies according to numerous factors, such as: age and nutritional status of the patient, presence of associated chronic diseases, extent of the wound, suture material, hemostasis, tissue trauma, and infection. Any one, or combination of these, may ultimately determine the fate of the wound. Recent experimental and clinical work has firmly established that the basic and essential requirements for normal healing of a wound are: (1) a state of positive nitrogen equilibrium; (2) a normal fluid and electrolyte balance, and (3) adequate concentration of vitamin C.

Protein is the basic constituent of protoplasm and consequently of all tissues of the body. During starvation, or upon a non-protein diet a negative nitrogen balance results which is manifested clinically by retardation in growth. Since wound healing is a biologic phenomenon conforming to laws of growth, any deficiency in the protein content of the body will affect the rate and extent of repair of tissue after injury. Abnormally low plasma protein has its specific affect by retarding the proliferation of the fibroblasts, thus prolonging the phase of fibroplasia. The plasma proteins also play an important role in the maintenance of normal fluid exchange between the blood and the intercellular tissues. Tissue edema, resulting from hypoproteinemia, seriously interferes with wound healing. The severity of the edema parallels the extent of plasma protein depletion; especially when the albumin factor is decreased to a greater proportion than the globulin. Thompson, *et al.*, have shown that abdominal wound disruption occurred in 72 per cent of dogs operated upon in the presence of hypoproteinemia. It has also been shown that edema of the stoma and of the intestinal wall after an anastomosis inhibits intestinal motility. This is probably due to reduction in the size of the lumen as a result of tissue swelling, and to the inability of the edematous muscular layer to contract. As *a priori*, the correction of the patient's nutritional and fluid balance, preoperatively and postoperatively, is imperative. If for any reason enteral administration is not feasible, protein can be given intravenously as whole blood, plasma, serum, or protein hydrolysate. The amount and choice of the infusion will depend upon the specific needs of the individual patient.

Of the vitamins, ascorbic acid is an essential requirement for the production and maintenance of intracellular cement which is composed largely of collagen. Wolfer, *et al.*, have shown that human subjects on prolonged ascorbic acid depletion exhibit an approximate 50 per cent diminution in tensile strength of healing wounds. These subjects also exhibited a recognizable lack of collagen and reticulum in their healing wounds.

In the aged, wounds heal more slowly and less firmly than in the young. The retardation is complicated by associated disorders which accompany people in this age group. Elective surgery may be performed safely on these patients providing the surgeon carefully evaluates the risk and effectively corrects any nutritional imbalance.

The utilization of sulfa drugs and penicillin to inhibit the growth of bacteria has been well established, however, they are not entirely effective in combating wound infection. The application of sound surgical principles is still the most important method of preventing infection. Local trauma, presence of necrotic tissue, foreign bodies, or blood clots invite infection. To avoid local deleterious

minimum of reaction. Bear in mind that a suture is a foreign body—hence, use the least amount and the smallest size consistent with its holding power over the tensile strength of the tissue to which it is applied. There is a great deal of controversy as to the choice of suture material. Until such time when the "ideal suture" has been found, the surgeon should depend upon his suture technique to minimize the deleterious effects of the suture material on healing. Whether one uses an absorbable or non-absorbable suture is immaterial, providing, every known factor regarding the vagaries of the suture material is known by the surgeon. To use a large size catgut when a smaller size will do is asking for complications. The same can be said of any suture.

Accurate approximation of the tissues and avoidance of "dead spaces" are essential for prompt healing. Approximation should be done without puckering, tension, or undue laxity. Wide separation of tissues creates "dead spaces" which become filled with exudates that may delay healing, or predispose to infection. When tissues are sutured under tension, the inevitable result is tearing and *strangulation. These complications are serious, especially when hollow organs are* involved. Finally, bear in mind to avoid suturing tissues of unlike histological structure.

HEMOSTASIS

The surgeon should make every effort to maintain hemostasis during an operation. Hemostasis should be produced concurrently with each operative step. For instance, when making an abdominal incision do not incise the fascia until you have stopped all bleeding points in the superficial layer; do not open the peritoneum until all muscular bleeders have been ligated. The peritoneum must never be opened until the entire wound is completely dry. This will eliminate the presence of numerous forceps, which not only traumatize the tissues, but also interfere with freedom of movement. Do not wait to ligate a large vessel until all bleeders have been clamped, because you may not be able to distinguish which hemostat is grasping the large vessel, and hence you may tie it insecurely. If possible, isolate, clamp, and ligate before dividing.

If, while operating, a large vessel such as the cystic artery has been accidentally severed, apply digital pressure; then, by releasing slowly, the bleeding point can be detected, grasped with a hemostat, and ligated. Avoid plunging blindly into a bleeding surface in an endeavor to control hemorrhage. The peritoneum must never be closed until inspection reveals that all bleeding and oozing has been controlled.

For inaccessible parts, where it is impossible to apply ligature, the bleeding vessel may be secured by packing with gauze. Packing should be removed in 24 to 48 hours, and if necessary, repacked. Gauze packing should be avoided in the peritoneal cavity as much as possible. It should be resorted to only when absolutely necessary, as it invites infection and is not a method of drainage. There is always the danger of secondary hemorrhage in osseous cavities, hollow viscera, intracranial sinuses, or abscess cavities. Petrolatum gauze is less irritating and less apt to adhere to the wound.

Valuable contributions have been made in the use of absorbable hemostatic agents by Ingraham and Bailey on fibrin foam, by Frantz on oxidized cellulose, by Correll and Wise, Pilcher and Meacham, Light and Prentice, and Jenkins and Janda on gelatin sponge or foam. The basic mechanism involved in the use of these

substances is to hasten the formation and give structural support to the clot. The use of oxidized cellulose and gelatin foam is of considerable interest to the general surgeon. They are of particular value in the control of hemorrhage where suturing is not feasible. They should entirely replace the cotton sponge as a pack to control bleeding in inaccessible parts, to control bleeding from the raw or damaged surface of the liver after cholecystectomy, and after splenectomy. According to Jenkins, gelatin foam is especially suitable to preserve the continuity of a vessel which one would not choose to ligate if there was any other alternative. There are innumerable conditions where an absorbable hemostatic agent may be utilized. The introduction of these substances is a definite advance in surgery which should enable one to perform more extensive surgery with greater safety and at times lend itself as a lifesaving measure.

Hot moist sponges are commonly used in the abdominal cavity to absorb secretions and oozing blood. When extremely hot, these irritate the peritoneum, causing excessive exudate and postoperative adhesions. During a gastro-intestinal operation sponging should be done with care, as roughness will traumatize the mucous membrane. The purpose of flexible intestinal clamps is to control the contents and to facilitate suturing and not to control bleeding. Hence, avoid tight clamping, as this may traumatize the mucosa. Avoid including the mesentery of the bowel.

INCISIONS

Adequate exposure is the primary objective of an incision. It is often the secret of a masterfully performed abdominal operation, yet, too often we have seen surgeons struggling laboriously through inadequately made incisions. An ill-advised or improperly made incision requires more time and adds to the operative risk. *It should be made sufficiently long and wide enough to carry out any procedure* in the immediate field and permit exploration of adjacent organs. Avoid a needlessly long incision, if a shorter one will suffice. Nevertheless, the importance of gaining free access to the underlying structures outweighs any consideration of additional length of the incision.

At times it is a difficult task to decide which is the best incision for a given operation. The surgeon should choose the simplest one which allows the greatest freedom of manipulation of the underlying structures, without producing undue destruction of anatomical and physiological lines. Whenever possible, incisions should be planned so that the various layers are incised according to the direction of their fibers. In this manner physiological restoration of these structures is more apt to occur.

QUESTIONNAIRE

1. What are the attributes of an ideal surgeon?
2. Discuss the status of the surgeon during the following periods:
    (a) Philosophical;
    (b) Anatomical;
    (c) Pathological;
    (d) Physiological.
3. Who is considered the father of modern anatomy?
4. Why is William Harvey considered as the precursor of modern physiology?
5. Who was the father of modern histology?
6. Who was the originator of pathological anatomy?
7. Who was the founder of cellular pathology?
8. When was the French Academy of Surgeons founded?

9. Who is known as England's father of surgery?
10. Who organized the first medical school in America? When? Where?
11. What contribution did Schleiden and Schwann make to medicine?
12. Who is the father of modern medicine?
13. Who introduced antisepsis in surgery?
14. When was ether anesthesia discovered and by whom?
15. Define—vitalistic theory. How did it influence medicine?
16. Give a list of publications and discoveries in the 19th century which started the modern scientific movement.
17. Who were the pioneer physiological surgeons in America?
18. Discuss the physiological trend in surgery.
19. Define—"making the patient safe for surgery."
20. Name and discuss the three major objectives of a surgical procedure.
21. Discuss the future role of physiology in surgery.
22. Discuss the anatomical trend in surgery.
23. What is meant by "living pathology."
24. Discuss the pathological trend in surgery.
25. Discuss the importance of "manual dexterity."
26. Briefly describe the three phases of wound healing.
27. [illegible] wound occur?
28. [illegible]
29. [illegible]
30. What basic factors are essential for normal healing of a wound?
31. What is meant by "atraumatic technique"?
32. List the common methods of producing trauma while operating.
33. How do these influence healing?
34. What is meant by "suture technique"?
35. What is meant by "dead spaces"?
36. What happens when tissues are sutured under tension?
37. Discuss the importance of hemostasis.
38. If a large vessel, such as the cystic, has been accidentally severed, how would you proceed to control it?
39. How would you control a bleeder in an inaccessible part?
40. Discuss the value and use of fibrin foam, oxidized cellulose, and gelatin foam.
41. What important objective should you have in mind when making an incision?
42. Discuss the importance of a "planned incision."

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Chapter 2

FLUID THERAPY IN SURGERY

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Physiology and Physiopathology of Body Fluids. A correct knowledge of the physiology of body fluids and careful consideration of the abnormal findings in each case, in the light of recent therapeutic trends in this intriguing field, is the only way to help surgical patients when restoration to normalcy is beyond the limits of spontaneous recovery. To insure a quantitative correction of abnormalities, avoiding interference with physiological compensatory functions and preventing exaggeration of already present alterations, should be the aim of the physician.

In referring to the electrolytes of body fluids, their concentrations will be expressed in terms of chemical equivalence (mEq./liter). When the osmotic properties are the main concern, we will refer to milliosmols per liter.

The intracellular compartment constitutes the most important part of the organism—the cell. The differentiated cell of superior organisms demands a constant environment to preserve its physicochemical stability. *Normal reaction, osmotic pressure, temperature and nutrition for the cells is provided by the fluid surrounding the cells—extracellular compartment.*

Table 1.—Normal Concentration of Electrolytes in Body Fluids (mEq./L.)

Extracellular				*Intracellular*			
Cations		*Anions*		*Cations*		*Anions*	
Na	142	Cl	101	K	150	HCO_3	10
K*	4.5	HCO_3	27	Na	10	Cl	2
Ca	5	SO_4	1.5	Mg	40	HPO_4	88
Mg	3	HPO_4	2			Protein	80
		Org. acids	2			SO_4	20
		Protein	16				

* *Lower plasma concentration of potassium in 100 normals (4.62 mEq. per liter) has been recently reported by Love.*[28]

Not all the electrolytes of the organism are concerned with fluid balance; determination of sodium specific activity in bone suggests that 60 per cent of the skeletal sodium may be metabolically inert, for it does not exchange with Na during the equilibration period.[24] The exchangeable potassium content is probably 5 per cent less than total body potassium, the difference being 30 per cent of the potassium of red cells, brain and bone.[12] The role of magnesium in disease and its possible participation in the production of symptoms in cases suffering from fluid disbalance is not clear. Magnesium is related to carbohydrate metabolism. Mean serum magnesium value was 1.82 ± .12 mEq./liter in normals, while it was low in diabetic acidosis (1.27) and cirrhosis (1.49) and high in diabetics with *renal impairment* (2.31).[45]

There are fixed normal values within somewhat narrow limits for the total volume of intra- and extracellular fluids and plasma, as well as for the individual concentration of each ion. However, certain values are more essential than others and are kept within narrower limits; the mechanisms that prevent alterations in these values have priority over the others, and their abnormalities frequently are corrected at the expense of less essential ones. Thus constant reaction (pH) and concentration of extracellular electrolytes is essential. The osmotic pressure of extracellular fluid, that is, its total osmolar concentration is equal to that of the intracellular compartment. Any osmolar alteration of extracellular fluids is compensated by a change in volume.

The concentration of base regulates the total osmolar value of extracellular fluid, *the loss of chloride being easily compensated by an increase in carbonic acid.* Under normal conditions, it is the job of the kidneys to maintain constant values for water and electrolytes, after a balance of intake and necessary losses through gastrointestinal tract, lungs and skin is made. Nevertheless, sudden changes may occur, even under normal circumstances, that cannot be coped with rapidly enough by the kidneys. Here a supplementary mechanism of adjustment has to enter into operation. Homeostatic mechanisms of mammals provide enough safety devices to maintain constant values for the fluids and electrolytes of the body.

Adrenocortical Pituitary Influences.—As far as is known today, there seem to be nervous centers that control the function of distal organs directly or through hypothalamic-pituitary apparatus for that end effect. A surgical operation produces in most patients a significant ACTH-like response.[41] This response lasts from one to three days, the intact hypothalamus seems to be necessary for the increased release of ACTH from the pituitary to occur following trauma. Electrolyte changes brought about by adrenocortical activity are accompanied by changes in renal flow and tubular reabsorption. Sodium and water are retained and potassium is lost.[31] Hume and Wittenstein[31] have shown that following operative trauma, the release by the pituitary of an increased amount of ACTH is dependent upon an intact nervous connection between the injured area and the brain. It is generally agreed that adrenal hormones promote sodium retention via the renal tubules, and that stress, or the administration of ACTH results in retention of salt. However, Daughaday and Mac Bryde,[18] after careful studies of urinary steroids during salt deprivation and administration of desoxycorticosterone and ACTH, conclude that maximum tabular reabsorption of sodium depends on an adrenal salt hormone, the release of which is independent of ACTH. The dosage of desoxycorticosterone that produced maximum sodium retention did not inhibit ACTH liberation, as evidenced by unchanged excretion of urinary 17-hydroxycorticoids and 17-ketosteroids Thus the secretion of steroids of the C-11 oxygen group is stimulated by ACTH while a separate salt hormone is excreted through an unknown mechanism. Burroughs *et al.*[b] isolated a very weakly androgenic compound in the urine of a patient with carcinoma of the adrenals, with marked salt retention. Dobriner[20] found that this salt retention hormone is present in small amounts in the urine of normal individuals. Finally, Grundy and associates[28a] using newer methods of chromatography and bioassay isolated a highly active salt retaining steroid, which was later crystalized and named aldosterone (because it differed from corticosterone only in that an aldehyde replaces a methyl group in the C^{18} position). Aldosterone, also known as electrocrotin, is at least twenty times stronger than desoxycorticosterone in its sodium retaining effect.

The water intake of the body depends on (*a*) water drunk, (*b*) water in the food and (*c*) water of oxidation. Water loss occurs through (*a*) lungs, as salt-free water; (*b*) skin, as a very diluted solution containing mainly sodium chloride; (*c*) gastro-intestinal tract, normally in small amounts; and (*d*) kidneys. The adjustments to normal or abnormal water intake and loss depend on the kidneys and on the water drunk under the demand of thirst. An excessive water load is taken care of by a decrease in pituitrin excretion. This causes a reduction in water reabsorption in the distal renal tubule with the consequent excretion of larger amounts of low concentrated urine. Water deficit is accompanied by an increase in thirst. If water is not available (actually, or indirectly, as in nausea and vomiting), post-pituitary secretion results in a water-sparing effect, the urine becoming scanty and concentrated.

A great proportion of body heat is lost through vaporization of so-called insensible water. The loss of water this way varies in direct proportion to body temperature, especially if the patient wears heavy clothing or covers; the relative humidity of the air is another important factor, and heavier water losses occur in extremes of summer and winter unless artificial temperature and humidity are provided through an air conditioning system. In winter, heated air becomes very low in relative humidity, provoking a great loss of water in the respiration, whereas on a hot summer day, a patient transpires copiously in a futile effort to increase loss of heat through evaporation, for part of the sweat falls out of the body, not serving the purpose for which it was produced. Just as with water, the kidney has the ability to conserve or excrete variable amounts of the body ions. From the average glomerular filtrate of 180 liters in twenty-four hours, about 179 liters of water are reabsorbed, and from the sodium, chloride, potassium, HPO_4 and SO_4 present in that filtrate, 99.6, 99.4, 93, 83 and 74 per cent, respectively are reabsorbed by the renal tubule. A very small variation in the reabsorption rate may result in marked changes in the total twenty-four-hour output of each of these ions.

Dehydration.—When dehydration is accompanied by starvation, which is almost always the case, loss of water and electrolytes occurs in both extracellular and intracellular compartments. Loss of potassium parallels that of nitrogen in simple starvation, without water deprivation. Lack of water increases the loss of potassium, but does not change nitrogen metabolism, whereas administration of water and glucose spares both. Dehydration due to loss of gastrointestinal fluid increases loss of potassium as compared with nitrogen, even if glucose, water and sodium chloride are given. Further derangement of potassium metabolism is produced in trauma, including surgery. Here, the increased adrenal cortical activity results in increased potassium loss. The potassium-nitrogen ratio is much higher than in starvation, probably due in part to direct action of C_{11-17} steroids on the kidneys and partly to the adrenaline effects on carbohydrate metabolism of the cell. The effect of adrenal steroids seems to be due to an increased glomerular filtration rate, not to tubular function. This second possibility is, however, suggested by the fact that tubular reabsorption of glucose has been found increased, while excretion of diodrast was not modified.[47] As to adrenaline action, it has been observed,[23] that potassium follows carbohydrate cycle from muscle to liver and vice versa. Potassium leaves the muscle together with lactic acid during exercise and shock. A similar potassium losing effect is produced by adrenaline.

During the period of negative balance that follows surgery, potassium leaves the cells and enters the extracellular compartment. Hyperpotassemia of sufficient

magnitude to cause symptoms occurs only in patients with renal failure, either primary or secondary to dehydration or shock. Similarly, in the course of diabetic acidosis, potassium and phosphate are lost from the cells; yet blood levels do not increase until circulation and renal function fail with dehydration[11]. Serum potassium values may also rise to high levels during fatal hyperthermia. Serum potassium changes produced by hyperthermia are basically similar, regardless of whether the hyperthermia is produced by external heat or by calorigenic drugs. Although serum potassium levels are sufficiently high to constitute the major cause of death under these conditions, other factors, particularly peripheral vascular collapse, may also be critical.[31] Ordinarily, conditions producing negative potassium balance give origin to an increased turnover of potassium without a noticeable rise in the blood level if the ability of the kidneys to excrete the ion is normal. Sodium is retained, and there is an increase in the extracellular fluid compartment which starts at the time of the operation and reaches its maximum about the second or third postoperative day. It amounts to about 2 to 3 liters.[27] At the same time, there is an increase in the renal excretion of potassium, probably due to the direct action of 17-desoxycorticosteroids, which prevents the elevation of blood potassium.[25] Potassium losses after surgery are greatest during the first twenty-four hours. It is lost in excess of nitrogen and is usually accompanied by alkalosis.[19]

Nitrogen loss following injury is proportional to the previous nutritional state; patients suffering from malnutrition at the time of operation lose less nitrogen, but the ones who enter the operating room without depletion recover faster and better, despite the larger loss.

Potassium loss is proportionally higher than nitrogen, that is, more than 2.7 mEq. of potassium are lost per gram of nitrogen, and the coefficient $\frac{K}{N}$ diminishes with the state of nutrition to approximate tissue composition in the completely depleted patient. Sodium is retained by the kidneys but water is retained in greater proportion; a paradoxical fall in plasma sodium is the logical result; blood sodium returns to normal during diuresis of convalescence, when proportionally more water than salt is lost and fluid volume and osmolar concentrations are stabilized.

During the first part of the postoperative period, while negative nitrogen and potassium balance exist, potassium administration would be of no avail unless the intracellular catabolism or protein-potassium could be reversed. It has been shown that only after very large amounts of amino acids are provided is it possible to produce a positive balance of nitrogen; this, however, is not considered advisable; no evidence of shortening of convalescence or clinical improvement has been obtained with this therapy, which is not devoid of danger. A negative potassium balance cannot be reversed by increasing blood levels, and the administration of potassium alone would only increase the renal load and endanger the life of the patient with resultant hyperkaliemia. *Unless the patient is losing potassium through gastrointestinal drainage where replacement fluid therapy should compensate for the loss of potassium as well as other electrolytes and water, there is no need for potassium in a normal postoperative case.* That post-traumatic or postoperative sodium and water retention occurs is now an incontrovertible fact. However, in the presence of complicating factors, special therapeutic uses of water and salts are necessary in each particular case. The postoperative patient who loses potassium in the proportion to nitrogen as is present in the cells and has enough urine volume to excrete it,

is comparable to a case of starvation except perhaps for a faster rate of catabolic process. As long as that proportion is maintained, he has no electrolyte disturbances to fear. Like a case of starvation, the postoperative patient shifts soon into a convalescent stage when the ACTH effect is gone, the cells are avid for nitrogen and potassium, and the kidney no longer retains sodium. If this period coincides with normal gastrointestinal function, and the patient can be fed, there is no need for complicated serum-electrolyte determinations or intravenous formulas. However, this is not always the case, and the supply of amino acid nitrogen in correct porportion to potassium, as well as water, glucose and sodium chloride could be indicated.

Cannon and associates,[9] found that animals that were given potassium chloride in addition to the basal ration which was deficient in potassium, grew at a greater rate. In parenteral feedings potassium should be added to amino acids and carbohydrates to meet the nutritional needs of the patients.

There is another aspect of the electrolyte problem—that concerned with carbohydrate metabolism—which should also be taken into consideration. Potassium leaves the cells with lactic acid during muscular contraction or whenever there is a breakdown of muscle glycogen. By the same token, the intake of potassium by the cells is increased when glucose is made available. According to Soskin, 0.3 mg. of potassium is taken by the cells for every gram of nitrogen, while 3 to 6 mg. of potassium accompany every gram of amino acid nitrogen during the anabolic period. There is also a loss of phosphate during the period of negative balance, and a gain of the ion that parallels that of potassium in the opposite stage.

Renal Function in Surgery.—Starvation and dehydration decrease renal plasma flow, glomerular filtration and clearance. Since the extracellular fluid compartment is quite elastic and absorbs easily great fluid changes, some patients may not show clinical symptoms of dehydration preoperatively and yet they may actually be on the edge of a metabolic cataclysm. According to Breed and Baxter[3] only certain renal function tests designed to determine light changes in renal function, such as clearance studies, would give enough information on the real status of the patient. Moreover, decrease in extracellular fluid may be deceiving: a doctor is easily misled into thinking a patient's blood is normal when a hematocrit is just showing hemoconcentration in a patient with a decreased red blood count.

Survival rate up to 90 per cent was obtained in anuric patients after giving protein-sparing glucose and restricting electrolytes and water to prevent a rise in potassium and/or pulmonary edema. Usually no electrolytes and only 700 cc. of water should be given until diuresis starts; then only as much electrolytes as necessary for replacement.[4]

DIAGNOSIS OF FLUID IMBALANCE

The symptoms and signs of fluids and electrolyte changes are varied and sometimes may be rather confusing. If we consider this chapter as divided into abnormalities of each of the components of body fluids, it would be easier to differentiate conditions due to single or multiple variations.

A. Abnormalities of body water
    1. Water Deficit
    2. Water Intoxication
B. Abnormalities of volume without changes in osmolor concentration
C. Changes in pH, potassium and calcium metabolism.

Abnormalities of Body Water.—The symptoms and signs due to changes in body water may be divided into those due to deficit and those due to excess. A deficit of water is manifested primarily by thirst. In addition, the output of urine decreases and its specific gravity increases; there is an increase in the hematocrit and in the concentration of plasma electrolytes; there is also a reduction in body weight.

Thirst is a sensation provoked by a rise in the concentration of electrolytes, primarily. A deficit of water results in thirst after the electrolyte concentration increases. Since the osmotic pressures of intra- and extracellular fluids are maintained equal, the concentration of electrolytes in the intracellular compartment may increase after some of its water shifts to relieve the hypertonic extracellular fluid. If the water deficit increases, changes in osmotic pressure and volume, as well as changes in the individual concentrations of electrolytes complicate the picture. The opposite situation, that is, an increase in body water, occurs clinically in so-called water intoxication when large amounts of water are drunk after profuse sweating, or during intravenous therapy, when excess of glucose solutions in water are administered. In this condition, there is also a water shift, but in the opposite direction: from the extracellular to the intracellular compartment. The urine output increases and the concentration of the urine decreases. There is a reduction in the hematocrit and plasma protein concentration, and an increase in body weight. If water intoxication is severe enough, edema of the cells may interfere with life. The symptoms of water intoxication are: headache, vomiting, diarrhea and "heat cramps." There is a progressive tachycardia and tachypnea, while the blood pressure falls progressively.

Abnormalities in Volume without Change in Osmolar Concentration.—The changes in volume of body fluids may or may not be associated with changes in the concentrations of the electrolytes.

A decrease in extracellular fluid due to a loss of water and electrolytes is usually absorbed by the great adaptability of the extracellular compartment without repercussion on the cells unless it is of such a degree as to produce shock. Then oliguria and anuria follow with the resultant electrolyte changes.

Symptoms and signs may be absent in mild decrease of extracellular fluid. The presenting symptoms are those of the existing disease (vomiting, diarrhea, fever and other symptoms of intestinal obstruction, fistulous drainage, etc.). As the loss of extracellular fluid progresses, there is a proportional reduction in body weight, the skin shows a change in turgor, the tongue becomes dry and the eyeballs soft and sunken. There is a fall in body temperature. The blood shows an increase in the hematocrit and protein concentration, but changes in the concentration of the electrolytes. Finally, shock may occur, with tachycardia, hypotension, oliguria and anuria.

An increase in the extracellular fluids due to a proportional increase in water and electrolytes is also well tolerated. As the extracellular fluid volume increases pitting edema appears, as well as hepatomegaly, and the picture of pulmonary congestion. There is hemodilution and signs and symptoms of increased blood volume: elevation of venous pressure, prolongation of circulation time, functional heart murmurs. If the increase of blood volume is great enough, heart failure may occur.

Changes in Reaction of Body Fluids.—The symptoms due to changes in acid base concentration of the plasma can be divided into those secondary to respiratory changes: respiratory acidosis or alkalosis; and those due to metabolic abnormalities: metabolic acidosis and alkalosis. In both groups the acid-base changes may be

compensated or uncompensated, depending upon whether the pH of the blood is or is not maintained within normal limits.

Respiratory Acidosis.—If the rate at which carbonic acid is released by the lungs diminishes (respiratory depression by morphine, pulmonary disease with decreased gaseous exchange), the tension of carbonic acid in the blood increases. This in turn tends to change the proportion of sodium bicarbonate, carbonic acid, and according to the Henderson Hasselbalch equation ($pH = 6.1 + LOG \frac{B \cdot HCO_3}{H \cdot HCO_3}$), the pH of the blood; when a compensatory increase in plasma bicarbonate occurs, the pH returns to normal: compensated acidosis.

Respiratory Alkalosis.—An increase in the output of carbon dioxide results in respiratory alkalosis. The changes in blood pH and bicarbonate are comparable but in opposite directions to those described under respiratory acidosis. Such a condition may occur in patients suffering severe pain, in cases of encephalitis and in hysteria.

Metabolic Acidosis and Alkalosis.—Whenever an increase or a decrease in the intake or output of sodium or chloride occurs independently of the other, or when there is an increase in organic acids of the blood, replacing carbonic acid from plasma bicarbonate, a metabolic alteration of acid base balance results. The emergency respiratory mechanism compensates rapidly and prevents changes in the pH of the blood. Later on, a renal compensation takes place, provided enough supply of water and electrolytes is available, and in the presence of a normally functioning kidney.

The most common causes of metabolic alkalosis and acidosis in surgical cases are: vomiting, diarrhea and loss of fluids in intestinal obstruction or fistula. The symptoms of acidosis and alkalosis are mainly due to central nervous system effect of H and OH ions, H being a depressant, while OH has an excitatory action. Thus, acidosis produces depression in the sensory modalities and reflexes, ending in stupor and coma; alkalosis produces excitability, increase in tendinous reflexes and eventually convulsions.

Changes in Potassium and Calcium Metabolism.—The symptoms of potassium retention are usually mixed with those of renal failure. The renal Tm of potassium is such that large amounts of the ion can be excreted, to prevent hyperpotassemia, when enough water is available, in the presence of normal kidneys. High blood levels of potassium result in symptoms of the gastrointestinal and cardiovascular systems: nausea and vomiting, intestinal colics, diarrhea and enterorrhagia; the cardiovascular changes are manifested in the electrocardiogram by A.V. block, disappearance of P waves, depressed S.T. segment and high T waves. If severe enough, hyperpotassemia provokes cardiac arrest in diastole.

Hypopotassemia is a common complication of severe fluid loss and acidosis. It is bound to occur in surgical patients when large amounts of fluids are lost from the G.I. tract, especially if replacement is intended with sodium chloride alone, in patients that rely on intravenous fluid replacement. Symptoms of potassium deficiency are anorexia, weakness and eventually paralysis, disorientation and even coma. The signs are, at first, hyperactive tendon reflexes and muscular twitching, followed by areflexia, vomiting and paralytic ileus. The heart sounds are distant. Electrocardiographic changes have been described: low voltage, flat T waves and marked depression of S.T. segments. However, Ljunggren *et al.*[34] observed

During the preoperative phase, and during the early postoperative period, the latter characterized by pituitary-adrenal responses to trauma lasting usually three days to a week, the patient's needs are different and in some aspects just the opposite of those of the days to follow. Sodium retention and negative nitrogen and potassium balance are the main characteristics of the early postoperative period. The supply of water and electrolytes to replace physiological and pathological losses is the main objective.

Nutrition Replacement.—Force feeding and intravenous amino acids do not accomplish positive nitrogen balance in the normal adult unless the patient is depleted or unless testosterone is added. It has been shown[41] that in the preoperative period, protein or intravenous amino acid nitrogen only produces a positive balance if more than 20 grams are given per day, and most of the nitrogen is excreted anyway; in the postoperative period it is even more difficult to obtain a positive balance; the intravenous administration of large quantities of protein hydrolysate does not appear to improve convalescence. The negative balance of calories and nitrogen is of little importance in such a short period, and except for the glucose administered with the water of replacement, calories and nitrogen are provided later on, during the positive-balance period of convalescence. One hundred grams of glucose is generally the correct amount to be administered during the first postoperative twenty-four hours. The loss of nitrogen and potassium is thus effectively reduced,[26] and ketosis is kept at a minimum. Water which would otherwise be needed for excretion of nitrogen, potassium and ketone acids is also spared. More than 100 grams of glucose does not seem to further significantly decrease the negative nitrogen balance.[42] Unless pathological loss of fluids exists to justify extra water and electrolyte administration, additional sugar *per se* is of no avail and may be harmful, for it may result in additional renal loss of fluids.

The intravenous administration of glucose should be slow if glycosuria is to be prevented; only 6 to 12 gms. can be utilized per hour[34] in an average adult. The recommended 100 gm. dose needs a three-hour infusion period to avoid renal loss of sugar.[31] To eliminate the long and inconvenient vein feeding, rapid infusion of invert sugar has been suggested. It is assumed that the same antiketogenic and protein-sparing effect is exerted.[43] Invert sugar is a hydrolyzed product of sucrose containing equal amounts of glucose and fructose. There are indications that the assimilation of fructose is faster than that of glucose, and it is claimed that the rapid utilization of invert sugar permits one to give 100 gm. in solution intravenously in one hour.[43]

The conditions which require treatment with parenteral fluids tend to provoke the development of Vitamin B Complex deficiency. Persistent vomiting from gastrointestinal obstruction and other intra-abdominal diseases frequently result in cessation of food intake. In such conditions, as well as in postoperative states, the administration of dextrose parenterally is supported by years of clinical success. The requirement of thiamine has been shown to increase proportionally to the intake of carbohydrates. The task of metabolizing dextrose may reduce or exhaust the reserves of Vitamin B. In patients who are depending only on intravenous glucose for their nutrition, provision should be made to compensate for the lack of vitamins of the B Complex. A method for determining the needs of each of these factors in surgical patients is not known, but it would seem appropriate to supply an amount at least equivalent to the daily requirement. For this purpose, any of

However, the volume of gastrointestinal secretions may decrease considerably during *starvation*. *Hildes et al.*[39] observed in a subject with complete pancreatic fistula that the twenty-four-hour volume of pancreatic juice decreased to about 300 cc. from a control volume of more than 1000 cc. Sodium and potassium concentration remained close to that of serum.

When extracellular fluid is lost into the tissues, as occurs in burns, trauma and venous thrombosis, it is not easy to determine the loss accurately. A stretcher-scale for the accurate weighing of bed-ridden patients described by Bluemle and Elkinston[1] may be helpful in fluid balance determinations. In burns, Evans[22] recommends 1 cc. of plasma and 1 cc. of saline per 1 per cent of body burn per kilogram of weight in the first twenty-four hours, and about one-half as much for the second twenty-four hours. Then the swelling begins to resolve and the fluids are reintegrated to the extracellular compartment. Fluid therapy then is contraindicated. In the case of an ileus in which fluid has not been drained, 2 to 3 liters of gastrointestinal secretions may be accumulated in the bowel and should be replaced. However, as the bowel recovers, the accumulated fluid is reabsorbed and may become a load to the extracellular compartment.[42]

The composition of gastrointestinal fluids is different for each portion of the system; *the total molar concentration being estimated to be the same as in plasma.*[3] However, the analysis of drainages from the gastrointestinal tract in surgical *patients has shown*[42] *that digestive fluids may be hypotonic. This was particularly* true for stomach secretions. The most hypotonic secretions were found in elderly patients with gastric anacidity. Intestinal drainage was slightly more than ⅔ isotonic, while bile and pancreatic juice approximated plasma concentration.

Gastric juice is lost in pyloric or high intestinal obstruction, losing chlorides in amounts far greater than sodium in patients with free hydrochloric acid. The acidity of the stomach varies from very high in peptic ulcer patients, to low or absent in certain patients with gastritis and cancer. The determination of the acid content of the gastric juice is such a simple method that a reasonable determination of the electrolyte mass lost from the stomach can be easily made. As a matter of fact, most surgical patients have had a gastric test before going to the operating room.

Acid gastric juice contains chloride in the form of hydrochloric acid (free acidity). In addition, *hydrochloric acid combines with protein and acid salts such as phos*phate and carbonate (total acidity). The balance of the total chlorides of the gastric juice is in the form of sodium and potassium salts. As the degree of acidity of the stomach decreases, the hydrogen ion is replaced mostly by sodium, although the potassium salt may vary as much as from .5 mEq. per liter of very acid juice, to 35 mEq. per liter when the acidity is low.[27] According to Lans and co-workers,[33] the loss of potassium as a result of vomiting or prolonged gastric suction may be as high as 6 to 8 gms. of potassium chloride (81 to 108 mEq. of potassium) per twenty-four hours. These authors recommend the addition of 3 gms. of potassium chloride *to a liter of isotonic* sodium chloride solution in these cases. As an example, ulcer patients with potassium depletion due to a restricted diet of long standing who are operated on without *preparation and who vomit, may develop* hypokalemic alkalosis, especially if saline is used postoperatively. Such patients are apathetic and weak, with a silent abdomen. Plasma chloride and potassium are low, while plasma bicarbonate and urea nitrogen are high. Four factors are necessary for the development of hypokalemic alkalosis: potassium deprivation, stress, chloride loss

normal. This is the so-called compensated metabolic acidosis, and is characterized by a diminution in the carbon dioxide combining power. The osmolar concentration, however, is not altered. Where sodium loss predominates over chloride loss, as with intestinal drainage, the diminution of the carbon dioxide combining power does not occur, or occurs late. If the loss of sodium is severe enough, it causes a fall in sodium concentration which results in a decrease in osmotic pressure of the extracellular fluid. Although any decrease in osmolar concentration of the extracellular fluid is minimized by compensatory excretion of water, there is also some transference of water to the cells until the osmotic pressure of the intra- and extracellular compartments is equalized. The sequence of events that may follow, that is, edema of the cells, loss of potassium from the cells, and hyperpotassemia, may produce irreparable damage. The predominant loss of sodium over chloride is larger in small bowel secretion (20 to 40 mEq.), bile (30 to 50 mEq.) and pancreatic juice (70 to 110 mEq.), respectively. The excess of sodium in these secretions is due to the replacement of chloride by carbonic acid. The concentration of sodium and potassium is rather constant, 120 to 140 mEq. of sodium and 5 to 15 mEq. of potassium, respectively. If a chemical determination of the fluids lost can be made, the preparation of a replacement solution of identical composition is ideal. A solution can be made with chlorides, sodium and potassium, and sixth-molar sodium lactate. The deficit of sodium is probably never greater than 12 milliliters of molar sodium lactate per kilogram of body weight or the equivalent amount of sodium bicarbonate (1 gm.).[17] Therefore, 70 milliliters per kilogram of body weight is the maximum dose of sixty-molar sodium lactate over a short period. The solution developed by Darrow called *potassium lactate* contains sodium and chloride in the ratio of extracellular fluids with an amount of potassium (27.8 mEq. per liter as potassium chloride) which is unlikely to raise the potassium blood levels if given slowly (over a period of four hours or more). When bile or pancreatic secretion is lost, an almost quantitative replacement solution can also be prepared even if no analysis of those fluids is made, since the electrolyte concentration of these secretions is fairly constant. In the majority of such patients, however, mixed intestinal secretions have to be replaced, and the chemical determination of such fluids is not only difficult, but is always impractical.

It has been said before that the sum of the cation values determines the osmolar concentration of plasma; when alkaline intestinal fluids escape from the body, the quantitative replacement of base is in order. A solution containing 10 to 20 mEq. of potassium and 130 to 140 mEq. of sodium provides nearly quantitative replacement. Chloride loss, on the other hand, may vary approximately from 50 to 130 mEq. It can be safely provided in a concentration of 100 mEq. per liter, and the rest of the anion supplied as lactate. Such a solution can be prepared easily with one ampoule of 20 mEq. of potassium chloride (1.49 gms. in 10 cc.); 300 cc. of $\frac{1}{6}$ molar sodium lactate, which would provide 50 mEq. of sodium; and 600 cc. of saline that provides a little over 90 mEq. of sodium and chloride. This solution is only slightly hypertonic. To avoid the mixing of solutions, Solution "D" referred to previously,[11] which is available, is suggested. Solution "D" contains 50 mEq. of lactate and 100 mEq. of chloride against 138 mEq. of sodium and 12 mEq. of potassium. Hartmann's *lactate-Ringer solution* is not recommended because it has only 28 mEq. of lactate and 4 mEq. of potassium.

Plasma Substitutes.—Shock resulting from trauma or hemorrhage does not respond to electrolyte or glucose solutions alone. Morrison, Lundy and Essex[38]

found 100 per cent mortality in rats and guinea pigs after experimental hemorrhagic shock was treated with isotonic saline solution, whereas under similar conditions, plasma and whole blood resulted in 90 to 100 per cent survival. The survival rates in guinea pigs were 50 per cent with Dextran, 60 per cent with Periston and 80 per cent with Plasmoid. In rats, after Dextran, 70 per cent survival; after Periston, 80 per cent; and after Plasmoid, 80 per cent. No deleterious effects or pathological changes were observed after prolonged intravenous administration of these substances.

More recently pressor agents have come into increasing use for the treatment of shock. When used intravenously, as in the case of norepinephrine[56], they should be given in 5 per cent glucose solution, and careful records should be maintained of volume of fluid administered. An ampul of 4 cc. of norepinephrine 0.1 per cent base is added to 1000 cc. of 5 per cent dextrose and water. This solution is injected intravenously, from 2 to 3 cc. per minute initially. Blood pressure response is observed and the rate of injection adjusted to maintain the desired blood pressure. If fluid restriction is indicated or if large amounts of norepinephrine are required, more concentrated solution should be used. To avoid an associated water load, the same 4 cc. or norepinephrine can be diluted in 500 or 250 cc. of glucose solution.

TABLE 2. OUTLINE OF DAILY FLUID REPLACEMENT

Physiological Losses	*10% Dextrose in Water*	*Saline*	*Sol. B or Ten-lonmade Sol. of NaCl, KCl, NH_4Cl*	*Saline Plus KCl, 3 gms per Liter*	*Tadsomade Sol. of NaCl, KCl and Lactate, Sol. G or Darrow's Potassium Lactate Sol.*
Normal Preoperative Normal Postoperative	1000 cc.				
Excess Perspiration (No need to change Clothing)	1200-2000 cc.				
Very Excessive Perspiration (Clothes very wet, had to be changed)	2000-3000 cc.	500-1000 cc.			
Pathological Losses					
Vomiting or Gastric Aspiration (acid gastric juice)			500-2500 cc.		
Vomiting or Gastric Aspiration (no free acid)				500-2500 cc.	
Intestinal Aspiration Ileus					500-3000 cc.
Biliary or Pancreatic Fistula					300-750 cc.

QUESTIONNAIRE

1. What is the normal concentration of [illegible]?
2. How is an osmolar [illegible]
3. What is the role of [illegible]

4. Discuss role of adrenocortical activity in electrolytic balance during surgery.
5. Do the adrenal glands promote retention of sodium?
6. What is the range of reaction of body fluids?
7. Discuss the changes that occur in the pH of the blood under pathological conditions.
8. What factors will alter the osmotic pressure?
9. Discuss the normal exchange of intracellular fluid and extracellular fluid.
10. What are the ways by which the water intake of the body is done?
11. How does water loss occur?
13. In dehydration, what happens to the electrolytes?
14. What is the effect of starvation and dehydration on blood potassium?
15. What other factors will cause increase or decrease in blood potassium?
16. Discuss the role of adrenal steroid on potassium metabolism.
17. What is the normal amount of potassium deposited in glycogen, and in muscle, per gram of nitrogen?
18. What conditions will cause hyperkaliemia of sufficient magnitude to cause symptoms?
19. Why is it dangerous to administer potassium during a period of negative potassium balance?
20. When is potassium therapy especially indicated?
21. Is potassium required in a normal postoperative case?
23. Discuss the role of renal function in surgery.
24. What are the symptoms and effects of water deficit?
25. What are the signs, symptoms and effects of a decrease and of an increase in extracellular fluids?
26. How is respiratory acidosis developed?
27. How is respiratory alkalosis developed?
28. What are the common causes of metabolic acidosis and alkalosis in the surgical patient?
29. What are the symptoms and signs of potassium retention?
30. How is it manifested by electrocardiogram?
31. What are the signs and symptoms of hypopotassemia?
32. What are the symptoms and signs of calcium deficit?
33. Discuss the broad principles of parenteral therapy for the surg[illegible]al patient.
34. [illegible]
35. [illegible]
36. [illegible]
37. Discuss the uses of vitamins in the surgical patient?
38. Discuss the need for replacement of physiological loss of fluid.
39. How [illegible] od from the gastrointestinal tract?
40. What [illegible] gastric secretion, bile, pancreatic jui[illegible]
41. What is the composition of gastrointestinal fluid at different levels?
42. How much potassium loss may result from vomiting or prolonged gastric suction?
43. What is the potassium dosage required for the treatment of the above loss?
44. What are the four factors necessary to the development of hypokalemic acidosis?
45. Discuss the development of alkalosis as a result of loss of gastric juices?
46. What are the effects from loss of intestinal juices?
47. Discuss the treatment of alkalosis and acidosis.
48. Discuss the use of plasma substitutes and blood in surgical conditions.

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Chapter 3

BLOOD TRANSFUSION

By Ernest Nora

INTRODUCTION

Blood transfusion for therapeutic purposes is mentioned in ancient and medieval literature. The early literature is vague and contradictory and, for this reason, there is a great deal of confusion as to the interpretation of transfusion. The indications for transfusion in early times were usually for giving vigor to the old. In medieval times the drinking of blood was an accepted practice. This is often confused with the transfusion of blood directly into the veins. In the 15th century faith in the efficacy of blood transfusions was shared by all. Royalty, aristocracy and the intelligentsia were transfused despite the hazards. The practice had gained such widespread acceptance in France that it became necessary to enact a federal law to prohibit the use of transfusion in the practice of medicine because of the ensuing danger and high mortality rate.

For nearly two hundred years following Harvey's announcement of his theory of the circulation of the blood in 1628, many transfusions were performed, which, in many instances, were followed by fatalities. This was due to the fact that the barber-surgeons were uninformed about three important and essential principles which are required for a successful transfusion.

(1) Prevention of agglutination and hemolysis—hemolysis and agglutination do not occur unless unlike bloods are mixed. This was not discovered until 1900.

(2) Prevention of coagulation of blood—The long search for methods to prevent coagulation during transfusion and also in blood vessel surgery was rewarded by many discoveries. One of the most important is the de-ionizing of calcium by sodium citrate which is the most widely used. Hustin in 1914 was the first to use sodium citrate to prevent the coagulation of blood by de-ionizing the blood calcium. It is now the most used ingredient to prevent coagulation of blood in transfusions and has been since 1915 when Agote and Lewisohn perfected the citrate technique. Oxalates are used for prevention of coagulation in blood chemistry, but not in blood transfusion.

For a long time coagulation and agglutination were not differentiated. When two unlike bloods are mixed, agglutination and subsequent hemolysis may result. Blood clots in the circulation may cause death to the patient. Clots can be prevented by defibrinization, de-ionizing the calcium, or by collecting the blood in a paraffin-lined tube as is done in collecting blood for transfusion.

A smooth surface coating like paraffin is also used to inhibit coagulation by preventing agglutination of blood platelets and thereby preventing the liberation of prothrombin. A paraffinized tube is used in the Kimpton-Brown-Percy method of blood transfusion described later in this chapter. Heparin also prevents agglutination and clot formation. Heparin is found normally in the liver. Dicoumarol inhibits prothrombin activity. Vitamin K plays an important rôle in prothrombin

formation. Defibrinization of blood is no longer used because of the injury to the blood cells and the toxic reaction of the defibrinated blood due to the breaking down of some of the red cells and the liberation of potassium or foreign protein reaction.

The mechanism of clotting can be explained as follows: Where there is a stoppage of blood, settling takes place in the vessels. The blood platelets stick to the surface of the vessels where there is a decreased flow or an injured endothelial lining of the vessels. Thrombokinase (thromboplastin) is liberated from the platelets. The thrombokinase liberated in the presence of calcium, initiates the process of coagulation. Proteolytic enzymes from the fixed tissues or polymorphonuclear leucocytes may also initiate the process of coagulation. Fibrinogen is always present in the blood, as well as calcium, to complete the process of coagulation if thrombin is formed. Thrombokinase acts on calcium and prothrombin to produce thrombin. Thrombin acts on fibrinogen to produce fibrin. Clotting is prevented in the vessels by anti-thrombokinase and heparin.

(3) Perfect asepsis could not be attained until the science of bacteriology was established. Before the time of Pasteur and Lister, infections were very common due to lack of knowledge as to how to control the invading organism, and asepsis, therefore, was not understood.

With Landsteiner's discovery in 1900 of three of the four blood groups, and with the discovery of the fourth group by Decastello and Sturli in 1901, reactions and fatalities were reduced to a minimum. Jansky discovered four blood groups in 1907, and three years later, Moss, unaware of the work of Jansky, reported four blood groups. Jansky's group 1 was named group 4 by Moss, and Jansky's group 4 was named group 1 by Moss. Groups 2 and 3 were identical in both nomenclatures. Landsteiner, however, used the letters AB, B, A, and O. Other difficulties were overcome by Landsteiner's and Wiener's discovery in 1937 of the Rh factors. After each new discovery it was thought that all problems had been solved, and some few were, but many more developed due to the discovery of both the Rh and Hr factors, some of which are still unsolved.

The 1000 per cent increase in blood transfusions in the past ten years attests to their popularity and value in clinical application and in therapeutic practice. There is an increase in use of blood from 3.4 pints per bed in 1947 to 6.17 pints from July 1953 to June 1954 as reported by M. F. Lesses and H. J. Banton in July 7, 1955, in the New England Journal of Medicine.

GENERAL INDICATIONS

Transfusion of whole blood (fresh or stored), plasma, serum, erythrocytes, or platelets, depends upon the specific needs of the patient as well as the availability or suitability of the elements to be transfused. Transfusion of whole blood offers the greatest general good but also involves more hazards because more elements are transfused which are capable of producing reactions. Red cells alone, for example, carry the agglutinogens which are the most frequent cause of reaction.

The first indication for transfusion is to restore the volume of circulating blood as quickly as feasible, meanwhile to supply oxygen and CO_2-carrying erythrocytes. The second indication is to give the recipient essential elements for coagulation, proteins, leucocytes, platelets, antibodies and hormones. The third indication is to give the patient protein which increases the osmotic pull and assists in excreting excessive body fluids. In cases of hypoproteinemia, for example, the increased

protein may relieve the edema by drawing fluids into the vascular tree from which it may be excreted by the kidney.

Hemorrhage.—*Hemorrhage is a loss of all the components of blood*, whereas in shock the cells remain in the vascular system. The treatment of choice is to restore the blood volume with fresh whole blood or plasma.

Loss of blood volume occurs in hemorrhage due to diseases such as pulmonary tuberculosis, peptic ulcer, ulcerative colitis, epistaxis, typhoid, ulceration of the bowel, infectious hepatitis, ectopic pregnancies, trauma from accidents or surgery, attempted homicides, and war injuries and toxicity.

Diminution of blood volume by hemorrhage or loss of tissue fluid decreases the blood pressure 10 mms. of Hg (mercury) per liter of fluid lost. Because of the decreased volume, the heart pumps 20 beats per minute faster to compensate for the decreased volume. This volume is quickly replenished from the extravascular tissue fluids which amount to 15 per cent of the body weight or about 11 liters. It is not uncommon to find 40 per cent of the blood made up of these fluids, two to four hours after a severe hemorrhage.

Lymphatic drainage also helps to maintain fluid equilibrium by draining tissue fluids into the veins. The pressure in the arteriolar end of the capillary is normally about 35 mms. of Hg. The pressure in the venules is about 9 mms. of Hg. The osmotic pull of serum-albumin is 22 mms. of Hg. *If the blood pressure in the arterioles drops below 22 mms. of Hg, the fluid no longer goes from the arterioles to the tissue spaces but goes back into the vascular system.* The low protein content of tissue fluids has a correspondingly low osmotic pull. The osmotic pressure of the tissue fluid varies from 2 to 9 mms. of Hg which is much less than the serum-albumin pull of 22 mms. of Hg found in the vascular tree. There will be a balancing of the fluids between the blood and the tissue-fluid pressure which comes to an equilibrium of about 12 mms. of Hg. It can thus be seen that pressure, volume and proper concentration of serum-albumin are all necessary to prevent water logging of tissues.

It is well to remember that 1 liter of blood will raise the hemoglobin 20 per cent in the average adult, whereas, 500 cc., the amount usually transfused at once, will raise the hemoglobin from 8 to 12 per cent in the average adult. A corresponding loss of 1 liter of blood will reduce the hemoglobin about 20 per cent.

Blood Diseases and Vascular Damages.—The next group of indications for transfusion is in cases of blood diseases and vascular damages. In aplastic anemia, life may be sustained by repeated transfusion. In depressed bone marrow function a few transfusions of 500 cc. each are often life-saving measures used until the toxic factor is removed. In beginning treatment of pernicious anemia, transfusion is sometimes given. Pernicious anemia, however, rarely requires transfusion as one week's adequate liver or B_{12} therapy is equivalent to 500 cc. of blood by transfusion. We have seen thrombocytopenic purpura patients respond to repeated transfusions of fresh whole blood and platelets where transfusion of stored blood was of little value. In hemolytic jaundice, transfusion is of questionable value. Blood or plasma, especially the former, is of great value in prothrombin deficiencies. Many erythroblastic infants are now saved by transfusions of Rh-negative blood of from 50 to 100 cc. per day for three to five days or *exchange transfusions*.

The two new drugs, heparin and Dicoumarol, frequently used in vascular disturbances, also require transfusion to combat complications that they may produce. It is imperative then to be prepared to transfuse whole blood to bleeding patients

treated with either heparin or Dicoumarol. Vitamin K is given in 65 mg. doses in hemorrhage due to dicoumarol therapy. Vitamin K should not be given in blood transfusions because it may produce emboli.

Reports indicate that hemophilic patients are benefited by the globulin AHG fraction of blood. The mechanism is not known but it may lessen the resistance of the blood platelets. It is known that blood platelets are hyperresistant in hemophilic patients.

Surgical Indications

The prophylactic use of blood before, during, and after surgery is practically routine in long, drawn out surgical procedures where there is much trauma involved, or where large amounts of blood are lost. Several liters of blood may be used during one operation. The blood should be crossmatched in duplicate or triplicate before transfusion with all the prospective donors and recipients. Surgeons frequently give 500 cc. of blood before a gastric resection, and another 500 cc. or more during the operation. After surgery one or more units of 500 cc. may be given.

It is well to give a transfusion to any surgical patient whose hemoglobin is less than 9 grams per 100 cc. or less than 3.5 million erythrocytes per cubic millimeter. If the patient goes into shock, plasma or blood substitutes may be given in preference to whole blood when hemoconcentration is present. It is unwise to be guided solely by the blood count or by the percentage of hemoglobin because there may be a physiological variation of 20 per cent in a day. Therefore the blood volume should be determined either by the Evans blue or isotope method.

SHOCK

Shock may be defined as an acute peripheral circulatory failure brought about by the loss of circulating fluids. Phemister and Blalock insist that there must be blood loss in shock, while Moon and others include syncope with hypotension in this category. Moon includes a phase of nearly every disease in his books on shock. In this manuscript the author shall deal only with factors related to hemorrhage, loss of plasma, and tissue fluids in restoration of blood volume, and increased capillary permeability in relation to blood volume. Hemorrhage, treated above, may produce shock with a pseudo-paradoxical hemoconcentration. Hemoconcentration means the relative increase in number of erythrocytes in a given volume of blood as measured by the hematocrit, or the red count, or both. The absorption of fluids from the tissue may mask the increase of red cells by simple concentration of the blood.

The simplest way to ascertain hemoconcentration is to measure the packed

blood containing an anticoagulant. Centrifuge for fifteen minutes to pack the cells at the bottom. The normal for women is usually 42 per cent of the blood volume, whereas for men it is usually 45 per cent.

Any real increase of concentration of cells without excessive perspiration or diarrhea may be hemoconcentration which may be due to shock or hemorrhage. If the hematocrit reading is maintained for two hours after hemorrhage there is a hemoconcentration because the tissue fluids will have diluted the blood. Hematocrit readings of 55 to 60 per cent are frequently one of the best indications of shock.

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The simplest way to ascertain hemoconcentration is to measure the packed erythrocytes in a hematocrit containing an anticoagulant such as potassium oxalate, heparin, or sodium citrate. Fill a small hematocrit tube to the 100 mm. mark with blood containing an anticoagulant. Centrifuge for fifteen minutes to pack the cells at the bottom. The normal for women is usually 42 per cent of the blood volume, whereas for men it is usually 45 per cent.

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The amount of plasma to be given in hemoconcentration is calculated as the difference between the actual reading and the normal for the patient, in terms of 100 cc. of plasma for each mm. above normal. For example, a reading of 55 per cent on the hematocrit would require 1,000 cc. of plasma because the difference between 45 and 55 per cent is 10, and for each mm. requires 100 cc. of plasma.

There is an increased capillary permeability in shock which allows the fluids to leave the vascular bed and to enter and mix with the tissue fluids that bathe the cells. This is due, in part, to the falling of the blood pressure in the arteriolar capillary below the osmotic pull of the serum albumin which is 22 mms. of Hg. Probably the most important factors are toxicity of the histamine-like substances, poisons from bacteria, foreign proteins and anoxia as the result of the failing circulation, each complementing one another to produce a vicious cycle. Some cases are made worse by the injudicious administration of electrolytes of sodium chloride or glucose. These fluids drown the tissues and prevent utilization of O_2 with resultant hypothermia which in turn contributes in producing a cold, clammy perspiration with oliguria, and perhaps anuria with azotemia, followed by coma. Regardless of the etiology, the treatment is the same as that prescribed above, unless there is a cardiac insufficiency as in an acute coronary thrombosis with pulmonary edema. In the case of hemorrhage, whole blood is the replacement of choice.

Shock is frequently prevented by transfusions of blood or plasma. As much as 40 per cent of the blood may be lost and recovery will occur if transfusion is given in time. Wiener states that transfusion in 85 per cent of the cases will give satisfactory results before shock has set in, whereas only 40 per cent of the cases do as well if transfusion is given after shock has taken place. Plasma ought to give better results if hemoconcentration has set in. A hematocrit test ought to be done to determine hemoconcentration if feasible, if not, a red cell count ought to be done. Hemoconcentration is one of the precursors of shock and can be treated with plasma. The absorption of the tissue fluids by the blood will tend to lower both the hematocrit reading and red cell count, as will also the transfusion of fluids.

See section on blood volume to prevent hypervolemia.

BLOOD FRACTIONS

If the red cell count is as high as before surgery, or if hemoconcentration has set in, more plasma should be given, especially if there has been some hemorrhage and absorption of fluids. Excessive perspiration may produce hemoconcentration.

Obstetrical emergencies require blood in haste. Plasma can be used pending the transfusion of whole blood. Burns, with their attendant loss of white blood (serum), require plasma or serum. One liter of plasma is given for each 10 per cent of the burned surface of the body area.

Many patients are benefited by plasma even in the nephrotic stages of glomerulonephritis where there is a hypoproteinemia. Now that serum-albumin levels are easily and frequently done, eye hemorrhages are often checked in cases of hypoproteinemia by the administration of plasma which restores the blood-serum-albumin level to normal and clears the edema due to the hypoproteinemia.

ALBUMIN

Albumin, when available, ought to give even better results than plasma for the following reasons:

1. It is the constituent that has the smaller molecule and is lost most frequently.
2. It has the greatest osmotic pull to attract fluids from the tissues into the vascular system as explained under the headings of hemorrhage and shock.
3. It acts more rapidly and in concentrations of 25 per cent or more; it will draw over 3 times its volume of water into the blood vessels in fifteen minutes.
4. It is usually prepared so that it contains only about one-seventh of the sodium in an equivalent amount of plasma.
5. The small amount given because of the concentration tends to prevent shock because of its speedy action in keeping the fluid in the vessels.

Indications for serum-albumin are to prevent shock quickly and to keep the tissues from being water logged by hypoproteinemia. The albumin concentrate is especially useful where there is a nephrosis or nephrotic phase of glomerulonephritis.

ERYTHROCYTES

The first indication for the use of erythrocytes is where the homologous blood type is not available. Erythrocytes are given along with plasma, and washed erythrocytes, group O, are suspended in plasma, thereby preventing many reactions. The second indication is in the case of carbon monoxide poisoning. The third indication is in the case of a neonatal erythroblastic infant when the washed Rh-negative maternal erythrocytes are used if Rh-negative blood is not available and the infant is rapidly becoming anemic and icteric.

BLOOD SUBSTITUTES AND PLASMA EXPANDERS

Nearly all the old blood plasma substitutes and expanders of the past fell into disuse when large numbers of patients were infected with homologous serum Hepatitis. Recently new blood expanders were introduced and now are being tried clinically. Dextran is now sold in a 6 per cent solution in isotonic sodium chloride. It is carefully fractionated, nonpyrogenic solution which has approximately the molecular size of serum albumin. It requires no refrigeration and is ready for immediate use as a plasma volume expander given intravenously. One liter of the infusion is supposed to increase the plasma volume from 15 to 20 per cent. According to Pirani, much of it is excreted in the first twenty-four hours, and nearly all of it is excreted in seven days.

The American solutions are not supposed to cause Rouleaux formation and do not interfere with compatibility tests. Mollison and Frank Hartman state that the Dextran they used produced Rouleaux formation and interfered with blood compatibility tests. It is therefore desirable to draw blood sample before infusion of Dextran solutions since the subject is controversial. The contraindications to Dextran may be listed as increased hemodilution producing hypervolemia and changes in the kidneys, lymph nodes and also in the lungs. The allergic responses are usually cared for by using antihistaminics or Epinephrine hydrochloride. It is very valuable in preventing shock, it is easily stored, very available and most important of all, it has the decided advantage over blood plasma in not producing serum Hepatitis.

Gelatin [illegible] per cent in physiological salt solution may be given with comparatively few reactions. The main disadvantage is its quick elimination.

Gum acacia was introduced by Starling before plasma was generally used.

Plasma is superior to Gum acacia and has practically replaced it except where plasma is not available. Gum acacia was used as is Dextran in a 6 per cent concentration isotonic salt solution. The osmotic pull is about equal to that of blood plasma or 22 mm. of mercury.

Under foods may be included amino acids which are sometimes used to build up the blood and may be considered along with blood substitutes. One liter of 5 per cent amino acid in 5 per cent glucose is given very slowly. This is equal to about 50 grams of amino acid protein lysate per liter. There are several types on the market, some made from blood, others from milk.

CONTRAINDICATIONS

Contraindications for blood transfusion are few. It is hazardous to give blood where it is contraindicated as in the following conditions:

1. Malignant hypertension—In malignant hypertension the added transfusion load may precipitate cardiac decompensation or a cerebral accident.
2. Congestive heart failure.—Blood is frequently withdrawn from the patient with acute congestive heart failure.
3. Leukemia without bleeding.—The heart muscle is weakened by the infiltration of the immature leukemic cells so that a transfusion overload may produce heart failure.
4. Severe dehydration.—There is usually hemoconcentration in severe dehydration. Fluids or plasma are indicated.
5. War gas poisoning.—The same applies to war gas poisoning as in dehydration.
6. Generalized carcinomatosis unless radiation therapy or surgery is to be done. —There is no real value in giving a transfusion as it may increase the pressure in the diseased vessel.
7. Congenital hemolytic anemia.—The regenerative power is from 50 to 70 per cent as seen in the reticulocyte counts. The increase is so rapid that transfusion is not necessary. It was found also that cells added just increase the hemolysis and the jaundice.

HAZARDS

Hemolytic reactions rank first in importance due to specific incompatibility between the bloods of the recipient and the donor. These are usually due to errors in grouping, or where double cross-matching is not done after each transfusion to determine the subgroups or the Rh factors. Group O blood has given reaction when given to groups A or B. It also gives reactions when given to group AB, if the titer is high.

With the exception of group anti-M, the M, N, MN, and P groups are not of sufficient importance to be treated in a surgical monograph because they produce few if any reactions. The cold and room temperature auto-agglutinins are not frequently provocative of reactions because blood is quickly heated to 37° body temperature. One of the most common hemolytic reactions caused by a nonspecific agent is due to blood which has been stored too long. Heating refrigerated blood may produce reactions.

The newly discovered blood types are Kell, Cellano, Duffy, Lewis, Levay, Jobbins, Lutheran, Kidd, Jay, Miltonberger and Guth. These may account for

many of the heretofore unexplained reactions. The following blood types may also be the cause of transfusion reaction (Anti-O and Anti-H). Anti-M and -MN or -P rarely cause reactions.

DISEASES

Malaria, syphilis, hepatitis, or other bacterial infections may be transmitted with blood or plasma. Hepatitis has frequently been caused by giving pooled plasma. Pooled plasma is the mixture of plasma from many different donors.

Foreign body reactions may be due to old blood, too vigorous agitation, bacteria in the salt solution, or, the most common, improperly cleaned tubing. Chemical changes from too soft glass are rarely encountered today. The liberation of potassium from the old or broken erythrocytes has been implied where vigorous shaking has taken place. Sulfonamides may produce non-specific reactions.

SYMPTOMS AND DIAGNOSIS OF REACTIONS

The reactions vary greatly from a slight fullness of the head to almost immediate death of the patient if incompatible blood is given.

During the transfusion of from 10 to 100 cc. of blood, the patient may complain of fullness of the head, precordial distress, tingling sensations, usually followed by a severe, sharp pain in the lower back. The patient is anxious, restless and dyspneic, with a cold, clammy skin, weak, rapid, thready pulse, and may be nauseated or vomit. The gums may ooze and bleeding from the skin punctures or uterus may result. The urine is smoky brown to black, but devoid of erythrocytes. If the patient survives the first phase, oliguria or anuria develops. The Van den Bergh icterus index is high, and the sclera and the skin are yellow. The destruction of erythrocytes begins in a few minutes and the urine may be normal after forty-eight hours. The degree of blood destruction depends upon the titer of the hemolysins in the blood of either the donor or recipient rather than the amount of blood injected.

The azotemia is due to renal failure. Recovery in the renal phase is about 50 per cent. The uremic stupor, with edema, convulsions and added purpura, all appear early, usually in a few days, and produce the lower nephron syndrome.

However, since the treatment of a hemolytic transfusion reaction differs from that of a non-hemolytic reaction (pyrogenic, allergic, etc.), the following methods tend to substantiate that a hemolytic reaction has occurred.

Immediately after the reaction: (1) Urine examination for hemoglobin, (2) Van den Bergh test, (3) Hemoglobin and red cell count, (4) Recheck of type and cross-match, including Coombs' test on original samples and on freshly drawn blood.

Twelve to twenty-four hours after reaction: (1) Van den Bergh repeated, (2) measurement of fluid intake and urine output, (3) observation of skin and eyes for jaundice, (4) hemoglobin and red cell count.

TREATMENT OF REACTIONS

First, ascertain the cause and, if mild, remove the cause and continue the transfusion. If a series of reactions has occurred and new tubing has been used, improper cleansing of tubing may be the cause. Many types of cellulose, plastic or rubber single-service tubing are now available.

Alkaline treatment of new rubber tubing may prevent many reactions, *i.e.*, soaking with NaOH for six hours before cleansing.

New glassware should be treated with sulfuric acid dichromate solution, then neutralized with tap water, followed by dipping in two changes of distilled water.

Any apparatus which has been sterilized more than ten days previous to transfusion should not be used unless it is resterilized. Sterilized apparatus kept sealed in an air-tight container may be used without resterilization.

To prevent allergic reactions the donor should fast. It is better to use donors who are not allergic to foods, drugs or horse serum as given in an antitoxin. Adrenalin .5 cc. frequently abates the symptoms of the reactions. Pyribenzamine or benadryl may be of help if given early or in advance where there is a history of allergy. Alkalinization with sodium bicarbonate is said to be of value in preventing reactions.

In cases of anuria a special routine should be followed. It may be summarized as follows: The patient is not to be given more than 1000 cc. of liquid per twenty-four hours with no electrolytes unless indicated. Bull's diet consists of dextrose 400 grams, 100 grams of peanut oil, enough acacia is added to emulsify the above in one liter of water. This is given directly into the stomach by way of a small nasal tube. This tube is to be left in twenty-four hours per day and the above suspension is given slowly. This will sustain the patient and give all the needed food and liquid requirements necessary. It is free from electrolytes and proteins. Electrocardiograms give a simple and quick rough index of the potassium. Daily sodium and potassium determinations should be done on all anuric patients. This can be quickly done with a photoelectric flame photometer. Electrolytes can be given if necessary. The above treatment ought to reduce mortality to zero level even in anuric patients.

By exercising great care in typing and crossmatching, and by the use of single-service tubing and hard sterile glassware, even the mild reactions in blood transfusion can be reduced to below 5 per cent.

BLOOD GROUPING TECHNIQUE

The test is usually performed on a glass slide.

Take a wax glass-marking pencil and draw two separate circles on the slide, each about 1 in diameter. Into one of these circles place 1 drop of anti-A blood grouping serum (using the handy dropper furnished with each bottle), and into the other circle place 1 drop of anti-B blood grouping serum. In each circle then place a drop of equal size of 2 per cent salt water suspension of the red blood cells being tested. Prick the finger or the lobe of the ear, either of which has been previously washed with alcohol and allowed to dry for one minute, using 1 large drop of blood and 2 cc. of physiological salt solution. This makes a good suspension of red cells for the blood grouping. With a toothpick mix the 2 drops in one circle, and with another toothpick, mix the 2 drops in the other circle. Carefully tilt the slide back and forth to help the reaction occur. The mixture is smooth and even at first. Within a minute, clumps of red blood cells may begin to appear in the mixture. After three minutes, clumping (agglutination) is visible very readily with the naked eye. Some technicians wait for as long as thirty minutes and re-examine for clumping. Most technicians examine the mixture under the microscope in addition to examination with the naked eye. Clumping, or its absence, is carefully observed and the blood group, preferably test tube controlled, is then determined as follows:

If clumping takes place with anti-A serum it is group A.

If clumping takes place with anti-B serum it is group B.

If clumping takes place with both anti-A and anti-B it is group AB.

If clumping does not take place in either anti-A or anti-B sera it is group O.

Group O blood contains no agglutinogens in the erythrocytes. The titer of the agglutinins is low in most cases, therefore, no clumping will take place. The agglutinins are diluted by the recipient's blood which further aids in the prevention of clumping.

COMPATIBILITY TEST

Collection of Blood for Comparability Test.—Label 2 large, dry test tubes of 10 cc. capacity or more, and 2 small test tubes, each containing 2 cc. of citrate saline solution (1.5 per cent citrate in 0.9 per cent NaCl.).

Draw about 5 cc. of blood from the vein of the donor and put 4 drops into the smaller test tube containing 2 cc. of the citrate salt solution and the balance in a larger, dry test tube. Allow the blood to clot so that the serum may be separated from it. After clotting, centrifuge the clotted blood to hasten the separation. Use the same procedure for the recipient's blood.

The compatibility test is done as follows: Ring the slide by making 2 separate circles with a wax pencil, 2 cms. each, on the left and right halves of the slide, if a double hanging drop slide is not available do a test tube check.

LEFT HAND SIDE OF SLIDE	RIGHT HAND SIDE OF SLIDE
1 drop of patient's serum	1 drop of donor's serum
1 drop of donor's corpuscles	1 drop of patient's corpuscles

Run in duplicate for double check. Stir with a toothpick and cover with a cover-glass. Examine at once and at the end of two and ten minutes. Re-examine after thirty minutes. Agglutination should not occur.

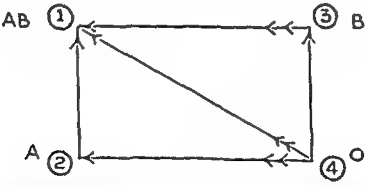

FIG. 1.—The arrows in the diagram all point to AB which is the universal recipient. The diagram also shows that Moss' group 4 and Jansky's group 1 or Landsteiner's group O blood can be given to any other group with comparitively little risk.

If agglutination occurs on the left side, with the patient's serum and the donor's cells, the blood cannot be used. If clumping, or agglutination, occurs on the right side, with the donor's ...

T

a ... occurs the transfusion is stopped at once. Measures are then taken to combat the reaction as mentioned in the section on treatment.

When incompatible blood is transfused the patient may have not only the injected erythrocytes destroyed, but an equal or added number of the recipient's cells may be destroyed by the serum of the donor. Transfusion of incompatible blood may cause clumping before it becomes hemolized. The clumping of cells in the vessels before their destruction by hemolysis may stop the blood-flow to the parts supplied by the vessel. The stoppage of the blood-flow may result in death to the part being supplied by the blocked vessel. The brain and kidney suffer the greatest damage, and for that reason, the symptoms referrable to kidney function are the most pronounced when transfused with incompatible blood. The liver and reticuloendothelial system give evidence of their overwork by the overproduction of bile and jaundice.

The table below illustrates the agglutinogens, the agglutinins and the Jansky and Moss classifications in relation to Landsteiner's (International) grouping.

Landsteiner	Agglutinogen in erythrocytes	Agglutinins in serum	Sera contains	Jansky	Moss *
Type O	None	ab	Anti-AB	1	4
Type A	A	b	Anti-B	2	2
Type B	B	a	Anti-A	3	3
Type AB	AB	None	None	4	1

From the above table it is obvious that Group O blood may produce violent reactions if the titer of ab agglutinins is high and group O blood is used as a universal donor. Group O blood contains agglutinins ab and if the titer is high considerable agglutination of the recipient's cells will take place. McGraw *et al.* estimate that 25 per cent of group O bloods give reaction when used as universal donor.

THE RH FACTORS

One of the most recent scientific discoveries which found general clinical application was the association of the Rh factors to blood transfusions and erythroblastosis fetalis. In 1937 Landsteiner and Wiener found that a new antibody was produced when the blood cells of the Rhesus Macacus monkey were injected into other animals. This is the reason it is designated as the Rh factor.

Levine, *et al.* confirmed the work of Ottenberg that there was a relationship between the Rh factor and erythroblastosis fetalis. They also postulated the theory of immunization of the mother by the fetus where the father was Rh-positive.

Only human serum is used at present because of the failure of animal serum to agglutinate the cells of newborn infants. At first the sera were obtained from animals or from women who had given birth to children with erythroblastosis fetalis. The sera are now obtained by repeated injections of Rh-positive cells into Rh-negative women beyond the child-bearing age.

The Rh factors are separate and distinct agglutinogens, agglutinins and conglutinins, and are not related to groups A, B, O, AB, M, N, MN and P. There are now over 12 known Rh related factors, the most important being Rh^0, Rh^1, Rh^2 antigens (agglutinogens) which react with rh^0, rh^1, rh^2, rh^1rh^2 in the serum. About 85 per cent of the Caucasian race, 6 per cent of the Negro race and less than 1 per cent of the Chinese race are Rh-positive to type Rh^0. About 70 per cent of the Caucasian race is Rh^1-positive and 30 per cent is Rh^2-positive.

Human erythrocytes contain 4 sets of genes in the chromosomes, one pair of genes for the A, B, AB, and O, a second pair for the M, N, and MN, a third set which contains 6 genes instead of only a pair for the Rh agglutinogens or their absence, and a fourth set which determines the presence or absence of the Hr factor.

The presence of any one or more of the dominant Rh-positive factors designates the patient as Rh-positive. The absence of these factors in the erythrocytes designates the patient as Rh-negative.

Rh-positive genes are dominant (Rh); the Rh-negative genes are recessive and are written rh genes. These rh genes are recessive and follow the Mendelian law as do all other blood groups. The Rh-negative individuals do not have the anti-Rh agglutinins in their blood serum unless they have had Rh-positive erythrocytes introduced into their blood stream. The introduction of Rh-positive erythrocytes into the blood stream of an Rh-negative individual stimulates antibody formation to destroy Rh-positive cells. This is what occurs in life to produce the hemolytic reactions when more than one transfusion of Rh-positive blood is given to an Rh-negative recipient. This is the same mechanism that produces erythroblastosis or hydrops fetalis in infants when the placenta permits the exchange of antibodies from an Rh-negative mother and her Rh-positive child.

The same mechanism applies to the Hr factors. It differs from the A, B, AB and O groups in that in all these groups there is normally an antagonism, as in group A for group B, and vice versa, as explained above. In the Rh and the Hr groups this antagonism has to be stimulated by an antigen which is the positive factor found in the erythrocyte. True group O does not carry an antigen (agglutinogen) but the serum has agglutinins a and b. Rh-negative serum does not normally have an agglutinin against the Rh-positive factors, whereas in the A, B, and AB, it is found without introduction of other red cells.

Any Rh-negative individual who receives Rh-positive blood transfusions may develop antibodies against the Rh factor. An Rh-negative mother may develop antibodies if the cells from her Rh-positive fetus enter the maternal circulation.

If the mother is transfused after being sensitized a hemolytic reaction results in about 50 per cent of the cases. In a personal communication, I. Davidson stated that he and Kurt Stern have demonstrated that injections of Rh positive into Rh negative men was approximately six times as likely to produce Rh antibodies when the blood was ABO compatible for the recipients, than when it was ABO incompatible. Antibodies C developed in approximately 60 per cent Rh negative men following injection of Rh, (CDe). When more than one transfusion of Rh-positive blood is given to an Rh-negative recipient, it produces hemolytic antibodies in nearly 50 per cent of those so transfused. An Rh-negative mother having an Rh-positive child whose antibodies pass the placental barriers may produce antibodies in the mother capable of causing hemolysis with Rh-positive cells and erythroblastosis or hydrops fetalis in the child. Sensitization means that the antigens, or Rh-positive cells, stimulate hemolytic antibodies in an Rh-negative individual. These antibodies produce reactions such as clumping of the cells and hemolysis in adults or erythroblastosis fetalis in the fetus of a pregnant, sensitized mother.

From the foregoing, it is obvious that Rh factor typing is necessary before transfusions in all those who are Rh-negative.

Anti-Rh^0 and anti-Rh'' are readily available today from many serum centers. Anti-Rh' may be obtained from Certified Blood Donor Service, Jamaica, New York and Chicago Serum Center, Mount Siani Hospital, Chicago.

THE Hr FACTOR

The pertinent information regarding the Hr factor may be summarized as follows:

The Rh factor accounts for only 90 per cent of the cases of erythroblastotic fetalis which is in turn dependent upon an Rh-negative mother having an Rh-positive fetus and a placenta which allows an interchange of agglutinogens and agglutinins between the mother and infant.

The discovery of the reciprocal relationship between the Rh and Hr by Levine, Weiner, Davidsohn, Potter, and Race, Taylor and Mourant, paved the way to a fuller understanding of the unsolved cases of erythroblastosis fetalis which are readily explained by the Hr and other factors.

Fifteen per cent of the Caucasians are Hr-positive and have the ability to immunize or sensitize nearly 40 per cent of the Hr-negative individuals.

Anti-Hr serum is obtained from Rh-positive mothers who gave birth to erythroblastotic infants, whereas, anti-Rh serum is obtained from Rh-negative mothers of erythroblastotic infants.

It naturally follows that the Hr factor is a separate factor which should be properly appraised. Besides being able to produce erythroblastosis in the infant, hemolytic reactions are produced in adults when Hr-positive blood is given more than once to an Hr-negative individual if the recipient produces anti-Hr agglutinins. The next logical conclusion is to test all donors and recipients for the Hr factors.

It was mentioned above that our supply of anti-Hr sera was obtained from Rh-positive mothers who have borne erythroblastotic infants. This means that they must have been Hr-negative and were immunized by Hr-positive blood to produce the anti-Hr antibody as in the case of the Rh sensitization described above in detail. Give only Rh-negative blood to Rh-negative patients when possible.

RH TYPING

We use the Diamond and Abelson methods, with a heated box-slide heater and use only human anti-sera as the animal sera proved unsatisfactory for typing infants as well as adults. The serum is fairly stable but frequently loses its ability to agglutinate after a few months even when stored in the refrigerator. Some dispensers recommend heating the viewing box to 45° to 50°C in spite of the fact that prozone reactions are more common at higher-than-room temperatures for periods of thirty minutes. Blood stored in a refrigerator for more than two days may give false negative reactions.

1. The view box is kept heated all day during work hours.
2. Place 2 large drops of fresh blood on a regular microscopic slide. Oxalated, heparinized or citrated blood may be used if the concentration of the cells is not less than 40 to 50 per cent of the total volume. Anemic blood may be oxalated with a dry oxalate to prevent clotting and centrifuged to concentrate cells.
3. Add 1 drop of anti-Rh_0 serum to 2 drops of blood, Anti-Rh^1 and Anti-Rh^{11}.
4. Mix and spread over the slide with a wooden applicator.
5. Place the slide in the groove on the heated glass plate of the viewing box and rotate back and forth until agglutination takes place which is usually less than one minute but may take ten minutes.

suspended red cells are only clumped if there are bivalent antibodies. Univalent antibodies or blocking antibodies are known as conglutinins, and they only act in the presence of serum albumin or other substances such as Dextran or with cells that have been treated with trypsin or papain. Most difficulties are caused by weak agglutinating sera which have a low titer and are frequently called negative, whereas they are positive when properly tested. Patients having a weak titer may have cells which provoke severe reactions when the red cells are injected into an Rh-negative individual. It is sometimes well, in doubtful cases, to test the blood at low, room, and body temperatures. For emergency work the usual Diamond and Abelson technique using 30 per cent albumin gives quite satisfactory results.

Two more groups ought to be mentioned. These are the Rh-negative male who receives transfusions of Rh-positive blood, becomes sensitized and develops hemolytic intravascular reactions; and the Rh-negative female in whose case extra precaution should be taken so that Rh-positive blood is not given. This is very important because the proportion is 6 to 1 that she will marry an Rh-positive husband, and if she becomes sensitized there is a 30 per cent likelihood that her children will develop erythroblastosis fetalis, icterus gravis or hydrops fetalis, all because of failure to do a few simple tests after the seventh month.

METHODS OF TRANSFUSION

Citrate Method.—The most commonly used method today is the collection of blood in bottles containing citrate solution for immediate use, or the use of blood stored in a bank and preserved with citrate dextrose. Silicon coated bags are now used for the preservation of blood platelets instead of the use of glass or paraffin tubes. Transfusions should be done within a matter of a few hours with a silicon bag and minutes in the case of a paraffinized tube. Silicon is said to distort red cells after a few hours.

The donor is placed on the table and a blood-pressure cuff is put on the arm. The donor set, which includes sedimentation vacuum bottle containing 70 cc. of 2.5 per cent sodium citrate in isotonic sodium chloride solution, is prepared. When the needle, 15 gauge, is in the vein, the vacuum is released and the blood flows into the bottle. The bottle is rotated so that the citrate solution is mixed with the blood. Five hundred cc. is usually taken.

Many hospitals now use vacuum bottles in which to draw blood which contains the standard citrate solution for 500 cc. of blood. If less than 500 cc. of blood is drawn in the bottle, the citrate will not prove a handicap as it is non-toxic and is quickly eliminated from the kidneys.

If more than 500 cc. of blood is given, it is imperative to have the donor rest for about thirty minutes. Milk, eggnogg, or orange juice are usually given to make up for the loss of fluid.

Citrate Technique.—Cleanse the forearm over any large vein with a good antiseptic. Apply a sphygmomanometer cuff high up on the arm and apply pressure to just above the diastolic reading. Insert a 15-gauge needle and collect the desired amount of blood, usually 500 cc. for an adult. The bottle usually contains 70 cc. of 2.5 per cent sodium citrate solution in isotonic sodium chloride solution, the same as is contained in the vacuum collecting bottle. Extra citrate, if present is quickly eliminated and is non-toxic. The patient is usually given saline solution just before the blood is given. The bottle containing the blood is either exchanged for the salt

solution bottle or is connected by a "Y" tube, using the same tubing as was used to administer the salt solution. The speed of the blood-flow is adjusted to the needs of the patient and the ability of the cardiovascular system to withstand the load at the rate of 2 to 4 cc. per minute.

In cases of shock 500 ml. of blood are transfused in about five minutes. The second 500 ml. can be given in ten minutes by forcing air into the bottle. Use either the Jorevelet pump, an atomizer rubber and bulb, a sphygmomanometer bulb or combination gravity pressure pump, and filter through sterile cotton into the bottle. It is better to force the blood indirectly into an artery to prevent vasospasm.

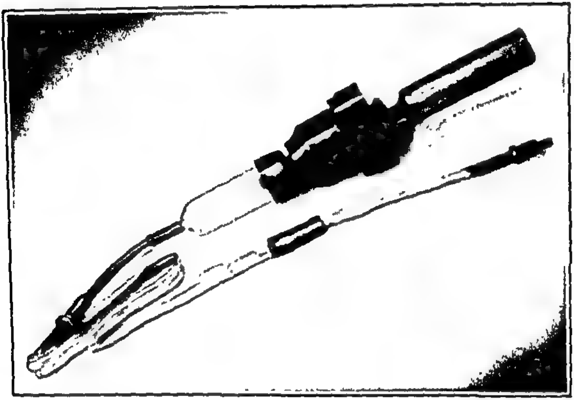

FIG. 2.—Single service tubing and filter.

Arterial transfusions are far superior to venous in cases of shock, thereby preventing anoxia of the brain which is the most delicate organ in the body. It also has a decided advantage in cases of cardiac arrest as eight minutes of cerebral anoxia usually causes death or a vegetative existence.

Single service tubing is preferable. Dr. Hartmann, the first user of cellulose tubing now prefers the single-service plastic tubing (see Fig. 2). This will prevent many foreign protein transfusion reactions which were due to faulty cleansing.

TREATMENT OF REACTIONS

First, ascertain the cause and, if mild, remove the cause and *continue the transfusion*. If a series of reactions has occurred and new tubing has been used, improper cleansing of tubing may be the cause. Many types of cellulose, plastic or rubber single-service tubing are now available.

Alkaline treatment of new rubber tubing may prevent many reactions, *i.e.* soaking with NaOH for six hours before cleansing.

New glassware should be treated with sulfuric acid dichromate solution, then neutralized with tap water, followed by dipping in 2 changes of distilled water.

Any apparatus which has been sterilized for more than ten days previous to transfusion should not be used unless it is resterilized in less than ten days.

To prevent allergic reactions the donor should fast. It is better to use donors who are not allergic to foods, drugs or horse serum as given in an antitoxin. Adrenalin .5 cc. frequently abates the symptoms of the reactions. Pyribenzamine or benadryl may be of help if given early, or in advance where there is a history of allergy. Alkalinization with sodium bicarbonate is said to be of value in preventing reactions. In the case of severe hemolytic reactions more compatible blood may be given and should be crossmatched in duplicate at body temperature.

Air embolism can easily be avoided with utmost care in clamping the tube before the bottle is completely empty.

In anuria, stimulation of diuresis by intravenous aminophyllin with dextrose solution may be of value. Decapsulation of the kidney is reported to have stimulated diuresis with recovery of the patient.

By exercising great care in typing and crossmatching, and by the use of single-service tubing, and hard, sterile glassware, even the mild reactions in blood transfusion can be reduced to below 5 per cent.

BLOOD BANKS

Blood banks serve to store blood in bottles rather than in the donor's vessels.

The advantages of a blood bank are apparent because of convenience, availability for surgical, medical and obstetrical emergencies, with replacement of the blood after the emergency has abated. The need of a long list of volunteer and professional donors is practically eliminated except for rare groups and group AB. The expense to the hospital is increased if the volume is less than 5 transfusions per day, except when type O blood is used as a universal donor this is not recommended.

Disadvantages of stored blood are:

1. Deterioration of blood after ten days with citrate, and after fourteen days citrate dextrose preservatives.
2. Shortage of skilled technicians seriously hampers the operation of an efficient blood bank.
3. Demands in a small hospital are so variable that it is not always possible to prevent waste and depletion of the blood bank.
4. Reaction due to cold blood in Raynaud's disease, paroxysmal hemoglobinuria hemolytic jaundice, and some severe anemias are common in stored blood.
5. Increased risk of bacterial contamination. Some few bacteria may develop at the low temperature at which blood is stored.
6. Donors who replace blood may not be as scrupulous as necessary in giving a history of past infections.
7. Prothrombin and platelets are decreased in stored blood.
8. Added mechanisms always give added risks. Changes in temperature controls may suffice to produce changes in stored blood to cause hemolytic reactions; it's use not recommended.

Most blood banks are now using the Fisher Nomenclature instead of the Wiener Rh notations. The equivalents are given below for comparison.

RH NOMENCLATURES

Wiener's Method Rh-Hr Notations	Fisher's Method CDE Notations
Anti-Rh_0	Anti-D
Anti-rh'	Anti-C
Anti-rh''	Anti-E
Anti-Rh_1	Anti-CD
Anti-Rh_2	Anti-DE
Anti-hr'	Anti-c
Anti-hr''	Anti-e

Extra Vascular or Cadaveric Blood.—The use of cadaver or blood recovered from the peritoneal cavity are both dangerous and not to be recommended. Even the filtering through gauze and the addition of citrate does not remove the toxic or possibly infected material therein contained. Cadaveric blood is blood stored in the vascular system of the dead. Peritoneal blood is blood found in the peritoneal cavity of the surgical patient.

UMBILICAL TRANSFUSION OF INFANTS WITH ERYTHROBLASTOSIS FETALIS OR HEMOLYTIC ANEMIA

Because of the early recognition of erythroblastosis fetalis and hemolytic anemia, transfusions are now frequently done within a few hours after birth, and for that reason, the umbilical route is to be preferred because of ease of operation which makes it unnecessary to cut down to the veins. The technique for umbilical transfusion is as follows:

1. Transfusion is usually done the first few hours of life where Rh-negative blood is indicated.
2. The indications for transfusion of Rh-negative or Hr-negative blood are where there is a history of erythroblastosis fetalis in the case of an Rh-negative mother and an Rh-positive infant with bilirubin over 15 mg. per cent.
3. If the mother has developed anti-Rh antibodies, the infant should be given a Rh test. If the infant is Rh-positive, or negative to only one or two groups, a typing should be done to rule out that the infant is heterozygous.
4. Davidsohn also uses Coombs' test for Rh antibodies before a transfusion.
5. An infant with jaundice, where no demonstrable antibody formation has been found in the mother, does not, as a rule, respond well to replacement blood therapy.

EXCHANGE TRANSFUSION

Weis/Gollin/Nora Modifications of the Standard Techniques

The use of exchange transfusion in erythroblastosis fetalis has two primary objectives: (1) To remove a high percentage of the baby's Rh-positive cells and Rh antibodies which it received from the maternal circulation, in order to minimize further hemolysis; and (2) to reduce the concentration of bilirubin in the infant's

serum. The effectiveness of this procedure can be *illustrated by the following* statistics from the Boston Lying-In Hospital, Beth Israel Hospital and Boston Children's Hospital. In 1945-46, prior to the use of exchange transfusion, the total recovery rate in erythroblastosis was 49 per cent, with kernicterus occurring in 33 per cent of all cases. However, by 1951-52, with exchange transfusion a well established procedure, the total recovery rate was 90 per cent with, kernicterus *occurring in only 1 per cent of all cases.*

Exchange transfusion is indicated under any of the following conditions, when the diagnosis of erythroblastosis has been established by clinical or serologic means. (1) In the infant under twenty-four hours of age where there is definite clinical evidence of erythroblastosis—jaundice, pallor, hepatosplenomegaly, petechiæ and a cord hemoglobin determination less than 15 gm. per cent. (2) Where there is an

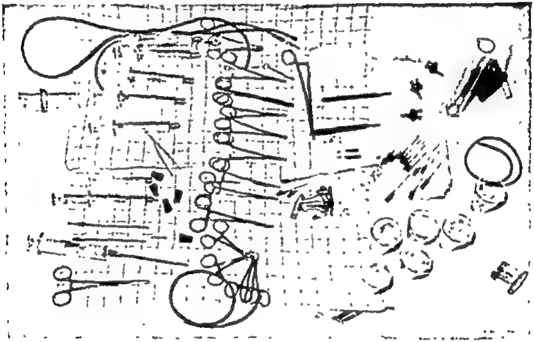

FIG. 3.—Instruments used in exchange transfusion setup.

unfavorable past history in previous pregnancies, such as kernicterus, stillbirths, or severe hemolytic disease. (3) In the immature infant, under thirty-eight weeks gestation, where there is clinical evidence of erythroblastosis. (4) Where the maternal antibody titer is 1:64 or higher. Repeat exchange transfusion is indicated when it appears that the serum bilirubin will rise higher than 15 mg. per cent in the twenty-four hours following the first. On the basis of experience, it would appear that a second exchange transfusion is indicated when the serum bilirubin continues rising above 15 mg. per cent during the twenty-four hours following the initial exchange.

The materials used in this procedure are as follows: A #6 woven umbilical cannula, two three-way stopcocks; rubber tubing; a recipient transfusion set; at least six 10 cc. or 20 cc Luer-Lok syringes; five metal basins; surgical instruments including scalpel, scissors, toothed forceps and mosquito forceps; umbilical cord tape; sterile sponges; sterile towels and drapes. Unsterile equipment should include oxygen, a heated mattress, blankets, bulb aspirator.

The most important requirement for a successful procedure is to have an efficient and smooth-working team of personnel. This exchange transfusion team should include six people: the operator, his assistant, a scrub nurse; an intern to observe the condition of the baby; a recorder; and a circulating nurse.

The exchange transfusion is carried out through the umbilical vein in the following manner. After sterile preparation of the abdomen, the umbilical cord is cut off about ½ inch from the skin margin. The umbilical vein is easily identified in the cross-section of the stump as a thin-walled, large vessel at the periphery of the cord, directly opposite from the two smaller, thick-walled umbilical arteries. The edge of the vein is then picked up with a small forceps and the umbilical cannula inserted into the vein. It should be inserted gently as far as it will go and until blood can be easily withdrawn. After the position where blood can be most easily withdrawn is found, the cannula is tied into place with the cord tape. The two stopcocks in series are then attached to the proximal end of the cannula. The proximal stopcock is connected to the donor bottle of blood and the distal stopcock to the rubber tubing leading to the waste blood basin. The metal basins on the sterile table should contain saline with 1:1000 heparin solution, used for rinsing the syringes. The operator begins the procedure by withdrawing 20 cc. of blood from the baby in order to create a slight initial deficit. He then alternately injects and withdraws 10 cc. or 20 cc. increments. While the procedure is being carried out, the condition of the baby must be constantly watched by an intern—checking the heart tones and rate almost constantly, checking the color and respirations, and aspirating the mucus from the pharynx, as well as administering the oxygen. This is most important. Restlessness and dyspnea indicate distress and usually mean that the injections and withdrawals must be slowed down. It is generally recommended that 1 cc. of 10 per cent calcium gluconate be injected following each 100 cc. of exchange. However, it may be necessary to inject the calcium more frequently, depending upon the infant's individual reaction to the citrate in the donor blood. A slowing heart rate or cardiac arrest is an indication for immediate injection of 1 to 2 cc. calcium gluconate. If the procedure goes well, 450 to 500 cc. should be exchanged, using Rh negative blood as the donor. However, if persistent signs of distress appear, it may be necessary to terminate the procedure sooner. Towards the end of the procedure the venous pressure should be checked with a piece of glass tubing connected to the stopcock. Normal pressure is 6 cm. of blood or less. If the pressure is normal and the baby originally had a moderate to severe anemia, an excess of 20 to 30 cc. blood may be injected. If the anemia was mild, equal amounts may be injected and withdrawn. If the venous pressure is elevated, a deficit of 30 to 40 cc. should be created. Repeated rinsing of the syringes with heparinized saline solution and the occasional flushing of the system with this solution will help prevent clots from forming and will facilitate smooth operation of the system. At the end of the procedure, the cannula is removed and the umbilical stump either tied or sutured. The stump should be kept moist so that a repeat exchange can be done through it if necessary.

Postoperatively the baby should receive prophylactic penicillin 150,000 u. once a day and vitamin K 5 mg. every eight hours for fifteen days. Feedings may be started in twenty-four hours. Serum bilirubin determinations should be carried out at least every twelve hours for the first forty-eight hours. The baby should be closely observed for rapidly progressive jaundice, lethargy and signs of central nervous system damage.

KIMPTON-BROWN-PERCY METHOD

In this method blood is prevented from coagulating by using a smooth vessel coated with paraffin. The principle of the method is to prevent the blood from coming in contact with glass, air, or rubber tubing. In this manner blood will remain uncoagulated for a period of eight to sixteen minutes.

The tubes employed for the collection of blood come in different sizes, the most common and convenient being a 700 cc. Percy tube. This tube is a glass cylinder, at the upper end of which a rubber tube is inserted. To this is connected a two-way valve for the purpose of causing pressure when the blood is being transfused, and suction when withdrawn. At the bottom of the cylinder is an S-shaped cannula which is a continuation of the cylinder. It tapers to fit any size vein. Before using, the tube is sterilized in an autoclave under a 15-pound pressure for twenty minutes and paraffinized with two ounces of grocer's wax. Great care must be taken to paraffinize the tube, otherwise clotting will take place.

Preparation of the Veins.—Under local anesthesia the veins of the donor and the recipient are exposed by an incision. A ligature is placed about the vein in the proximal portion of the donor, and in the distal end of the recipient. A bull-dog clamp is placed around the portion of the vein farthest away from the ligature in each patient. An incision is now made through all the coats of the veins midway between the clamp and the ligature. The flaps thus formed are held apart with small artery clamps. The donor's and the recipient's arms should be prepared at the same time.

As soon as the veins have been prepared, about 25 cc. of liquid paraffin is sucked into the Percy tube. The cannula of the tube is placed within the vein of the donor, pointing in the direction of the hand. The bull-dog clamp is removed, and by means of suction on the tube and pressure on the arm, blood is drawn into the tube. The liquid paraffin rises above the column, hence preventing air from coming into contact with blood.

When the desired amount of blood is drawn, the cannula is removed from the donor and inserted into the vein of the recipient. At this time the cannula is inserted so that it points toward the shoulder. The blood is made to flow slowly at first, and after the first 100 cc. it can be injected freely. If necessary, pressure can be used with the bulb attached to the rubber hose to allow the blood to flow as fast as desired. When the tube is emptied, the veins of both the recipient and donor are ligated, and the skin is closed with one or two interrupted dermal sutures.

With this method about eight minutes are allowed for the complete transfusion. Four minutes are allowed for the withdrawal, and four or more minutes for the injection. When whole blood is indicated, this method offers the most satisfactory results. The objection can be made that the veins are destroyed and that blood is given too rapidly causing heart strain in debilitated hearts.

QUESTIONNAIRE

1. Discuss the early history of blood transfusion
2. What are the three essential basic factors for a successful transfusion?
3. Who was the first to use sodium citrate to prevent coagulation of blood?
4. Describe the mechanism of blood clotting
5. Who was the first to announce the discovery of the various blood groups?
6. Discuss the general indications for blood transfusion.

Chapter 4

PRINCIPLES OF ANESTHESIA

By Kenneth S. Weiss

General Considerations.—Of the great strides modern medicine has made in recent years, the relatively new field of anesthesiology has contributed greatly to the safety and comfort of the patient. Whereas in the past much of the mortality and morbidity of surgery was a result of improper understanding of the problems of anesthesia, today the patient is immeasurably benefited from the advantages the specialty has made possible. Many of the surgical procedures that were considered impossible in the past have become commonplace as a result of the training and skill of the newest member of the surgical team. His scientific contributions to such fields as oxygen therapy, parenteral therapy, bronchoscopy, and diagnostic and therapeutic nerve blocks have made possible many of the recent surgical advances. With the introduction of the ultra-short acting barbiturates the induction of anesthesia has been made very pleasant and the use of the curare and curare-like drugs has made possible ideal operating conditions without greatly upsetting the patient's physiological mechanisms. Today, any patient who finds it necessary to undergo a surgical procedure may be assured that his sleep will be guarded by the best the field of anesthesiology has to offer.

Pre-Anesthetic Preparation.—Every patient who is to undergo surgery should be visited preoperatively by the person who will administer the anesthetic. Many of these patients have a greater fear of anesthesia than they have of surgery and a few words of assurance by the anesthetist will help to allay much of their apprehension. Some patients have never been in a hospital before and have a great fear of the unknown. By answering any of their questions regarding the proposed surgery, type of anesthesia, etc., the anesthetist can gain the patient's confidence and reduce many of his fears and worries. Nowadays, most of the public has knowledge of the use of intravenous anesthetic agents and by assuring the patient that he will be put to sleep quickly and painlessly he will come to the operating room in a more cheerful frame of mind. A patient with the right mental attitude and proper premedication will definitely facilitate the work of the anesthetist and that is why a few minutes chat with the patient prior to surgery is time well spent. While the anesthetist is visiting the patient he may find many salient points that can help the conduct of the anesthesia. He can learn something of the patient's temperament, his habits concerning drugs, alcohol, and smoking and whether he has any allergies or idiosyncrasies to certain drugs.

A superficial glance will give an idea of the patient's anatomy concerning his jaw, nose, teeth, superficial veins, posture, and type of body build. It is important to determine if the patient is a good, fair, or poor risk and will be able to tolerate a general anesthetic. If possible, and time permits, a physical examination should be made especially of the cardiovascular and respiratory systems. If this cannot be done, an inspection of the hospital record concerning the patient's history and

In the event that the dose of the opiate drug should be too large and the patient's respiration becomes depressed N-Allylnormorphine may be used to counteract the respiratory depressant effects. This drug antagonizes such drugs as morphine, meperdine and methadone. The usual dose is five to ten milligrams given intravenously.

Anesthesia for Head and Neck Surgery.—The anesthetic problems concerned with surgery about the head and neck are distinct from other types of surgical procedures. The main problem the anesthetist has to contend with is one of maintaining a satisfactory airway, without interfering with the progress of surgery. Many surgeons prefer the use of cautery for hemostasis, hence the choice of drugs that may be used is limited to the nonexplosive agents. Fortunately, however, none of the procedures require relaxation or profound anesthesia, so that only minimal amounts of drugs may be used to provide adequate light planes of anesthesia. Furthermore, a smooth induction without an excitement phase and an early return to consciousness following surgery without nausea or retching contribute greatly to reducing the postoperative morbidity of these cases.

For surgery of a minor nature, local infiltration anesthesia using one per cent procaine is probably the method of choice. The skin of the scalp and face is highly vascular and for this reason an anesthetic agent becomes quickly absorbed, hence the least toxic drug should be used.

If the patient is to be put to sleep an endotracheal airway is practically mandatory. No other technique enables the anesthetist to maintain an unobstructed airway and at the same time be removed from the operative site. Intubation may be performed in one of two ways: (1) With direct vision by means of a laryngoscope, the tube is introduced either through the nose or mouth; (2) "blind" technique of Magill, in which no laryngoscope is used. In many cases of head and neck surgery, the mouth cannot be opened or opened only with great difficulty and the "blind" method becomes mandatory. Using topical anesthesia, these patients should be intubated while they are awake because if they are put to sleep before being intubated, their airway may become obstructed; if the mouth cannot be opened they may asphyxiate before their respiratory difficulty can be corrected.

The variety of anesthetic agents that may be used for head and neck surgery is rather limited. Since a cautery is frequently used, explosive agents such as cyclopropane, ether and oxygen mixture, and ethylene must not be used. Induction is best accomplished by means of Sodium Pentothal which may be given as a 2.5 per cent solution or as a 0.5 per cent solution in a continuous intravenous drip. The patient is given sufficient Pentothal to produce sleep but not enough to depress the respiration. Following this, 20 to 40 mgms. of succinylcholine is injected through the intravenous tubing and when it has reached its maximum effect the cords are visualized and sprayed with a topical agent such as 2 per cent Pontocaine or 5 per cent Cyclaine and the patient is then intubated. Nitrous oxide in a ratio of three liters to one liter of oxygen is then administered through a semi-closed carbon dioxide absorption system. Pentothal may be given as an adjuvant to the nitrous oxide. If it is not desirable to administer an intravenous agent after the patient has been intubated, small amounts of trichlorethylene may be added to the nitrous oxide-oxygen mixture to increase its potency. If this method is used, a non-rebreathing valve close to the endotracheal tube for elimination of carbon dioxide is mandatory because of the toxic products formed when trichlorethylene comes in contact with the contents of the carbon dioxide absorber.

the operating table demand special considerations by the anesthetist. Patients who must be placed in the lateral position, such as for renal surgery, spinal or endotracheal general anesthesia may be used. In these cases spinal anesthesia may be performed unilaterally so that only the operative site is anesthetized, thus minimizing circulatory and respiratory complications. When general anesthesia is utilized, the patient is placed in the supine position for induction and intubation, after which he is placed in the desired position. It is mandatory to intubate all patients who are placed in the lateral positions.

For surgery of the abdominal wall, anesthesia may be produced by local infiltration of the area or by light general anesthesia consisting of nitrous oxide supplemented with Pentothal. Curare or curare-like drugs may be used if relaxation is necessary.

Transurethral procedures are best done under spinal anesthesia, unless for some reason, it is contraindicated. Nitrous oxide supplemented with Pentothal may also be used as an alternative. Because the cautery is used in transurethral surgery, only nonexplosive anesthesia techniques are employed.

Rectal surgery may be done under local, caudal, saddle block or general anesthesia. Regional anesthesia is ideal for the patients in the jackknife position. It causes perfect relaxation of the rectal sphincter and the airway is not an anesthetic problem. If the lithotomy position is used, the patient is induced with Pentothal followed by a general anesthetic agent such as cyclopropane. A small amount of curare may be used to relax the anal sphincter.

Perineal operations may be done under caudal, saddle block or light general anesthesia consisting of nitrous oxide supplemented with Pentothal. These procedures, as a rule, require a minimum amount of anesthesia and the method of choice depends upon the surgeon and the anesthetist.

For surgery of the upper extremities, anesthesia may be obtained by either regional or general anesthesia. Blocking the brachial plexus as it crosses the first rib will give excellent results. Nitrous oxide with Pentothal as a supplement may also be used if this is desired.

For surgery of the lower extremities, a unilateral spinal anesthesia confined to the operative site may be employed, or a general anesthesia may be used if desired.

Anesthetic for Chest Surgery.—Anesthetic procedures for chest surgery may be divided into two categories: (1) for extra-pleural procedures, and (2) for intrapleural procedures. Extra-pleural surgical procedures involving only the chest wall may be done under local infiltration, or intercostal block combined with local infiltration. For simple mastectomy or biopsy of the breast, light general anesthesia is probably the best for the average patient. Local infiltration causes a great deal of edema and at times renders a biopsy difficult.

For intra-thoracic surgical procedures it is mandatory that a general anesthesia be administered through a cuffed endotracheal tube. Induction may be produced by Pentothal followed by cyclopropane exclusively or supplemented with ether. Cyclopropane is a nonirritating gas permitting a rapid induction and rapid recovery after the termination of the operation, while during surgery it enables the anesthetist to control respiration with ease Ether has a wider margin of safety and will cause less cardiac irregularities. Ether is irritating to the respiratory tract when administered by means of insufflation, however, this is not a factor to be considered when a closed carbon dioxide absorption system is used.

The maintenance of adequate oxygenation and carbon dioxide elimination is undoubtedly the chief problem and concern of the anesthetist during intra-thoracic

surgery. When the pleural cavity is opened the intra-thoracic pressure becomes equalized with the atmospheric pressure and unless corrected the lung of the opened side will collapse and at the same time there is a shifting movement of the mediastinum. During inspiration the mediastinum shifts to the unaffected side and during expiration to the opposite side with the result that there is a continuous swaying back and forth which impedes adequate ventilation. This condition may be corrected by instituting positive pressure respiration and maintaining a small degree of continuous positive pressure, thus preventing the mediastinum from shifting. Controlled respiration may be brought about by hyperventilation to reduce the carbon dioxide content of the blood combined with inhibition of the Hering-Breuer reflex, or pression of the respiratory center with use of drugs. When the patient is in a state of apnea, the breathing is then carried out by intermittent manual compression of the breathing bag at a rate of 20 to 30 times per minute. In this manner the respiratory movements are controlled by the anesthetist thus providing ideal working conditions for the surgeon since the movements of the diaphragm are eliminated and at the same time adequate oxygenation is assured. An alternative method is the "supplemental" or "compensated" respiration which permits normal breathing by the patient aided by compression of the breathing bag at a pressure of 8 to 12 millimeters of mercury pressure to compensate for the inadequacy of the patient's respiratory efforts due to the pneumothorax on the surgical side.

Positive pressure should only be applied intermittently since continuous pressure of more than 3 millimiters of mercury pressure will interfere with the filling of the right side of the heart thereby lowering the blood pressure. The pressure should not exceed 20 millimeters of mercury pressure. Greater force may rupture the alveoli or may push secretions into smaller bronchioles thus causing areas of atelectasis and pneumonia.

In operations for bronchiectasis, lung abscess, or tuberculosis, the possibility of spillage of the infected material into the opposite lung is ever present. To avoid this complication a long cuffed endotracheal tube may be inserted into the bronchus of the normal lung and by inflating the cuff the bronchus is occluded thus preventing the infected material from entering the normal lung. Another method to avoid this complication is to place the patient in the supine or Overholt position.

Re-expansion of the lungs should be done about every half-hour and at the termination of the operative procedure. Before doing this all secretions should be aspirated otherwise they may be forced into the smaller bronchioles. The re-expansion should be done gradually and each succeeding respiration should further expand the lungs until they are completely ærated. By re-inflating the lungs the atelectatic areas are ærated and the patient's oxygenation and carbon dioxide elimination is improved. If part of the lung remains atelectatic, gentle massage of the affected area will permit the gasses to enter the atelectatic area. After the lung has been ærated several times, it is allowed to collapse and the operation may then proceed.

Before the chest wall is closed a long-acting local anesthetic, such as Efocaine or Eucopine in oil, may be injected into the intercostal nerves. This gives the patient considerable relief from pain thus reducing the amount of drug which would otherwise be required. It will also enable the patient to breathe more effectively and avoids respiratory depression due to large doses of narcotics.

After the surgery has been completed, a thorough aspiration of the tracheobronchial tree is done and the endotracheal tube is withdrawn. The patient is re-

turned to the ward or recovery room and placed in an oxygen tent or the oxygen may be given through a nasal catheter. The bed is placed in a ten-degree Trendelenburg position to promote postural drainage until the patient is completely awake and has a good substantial cough reflex.

QUESTIONNAIRE

1. What are the contributions made by anesthesiology?
2. Why should the anesthesiologist visit the patient preoperatively?
3. What are the purposes of the premedication drugs?
4. Which are the most commonly used sedation drugs?
5. Why should the barbiturates be given two hours before surgery and one hour before the opiate?
6. What are the physiological effects and advantages and disadvantages of the following opiates? Morphine, Pantopon, Demerol, meperidine.
7. What is the rationale for the use of Belladonna and scopolamine hydrobromide? Give dosage of each.
8 What drug and dosage is used to counteract overdosage of morphine?
9 What are the problems confronting the anesthesiologist in head and neck surgery?
10. Why is endotracheal anesthesia mandatory in head and neck surgery?
11. Give two methods of intubation; indications for each.
12 Why are explosive anesthetic agents avoided in head and neck surgery?
13 At the termination of the operation why should the endotracheal tube be left in place until the patient is conscious?
14 What other postoperative procedures are carried in head and neck surgery?
15. What are the problems confronting the anesthesiologist in intra-abdominal surgery?
16. How is regional anesthesia obtained for the abdominal wall?
17. How is visceral pain eliminated when regional anesthesia is utilized?
18. [illegible] ock?
19. [illegible] anesthesia.
20. [illegible] hesia?
21. [illegible]
22. What are the advantages in the use of nitrous oxide?
23. What are the advantages of an endotracheal tube in upper abdominal surgery?
24. Discuss the advantages and disadvantages of spinal anesthesia.
25 What are the commonly used drugs in spinal anesthesia?
26. What are the complications of spinal anesthesia during surgery that must be watched?
27. How would you treat hypotension?
28 How would you treat complete apnea?
29. What are advantages of continuous spinal anesthesia?
30 When is it especially indicated?
31 What anesthetic techniques are used for the following operative procedure: renal, transurethral, rectal, perineal, upper and lower extremities, extrapleural, simple mastectomy.
32. For intra-thoracic procedures what is the mandatory requirement to produce general anesthesia?
33 What are the advantages of cyclopropane in chest surgery?
34 What are the advantages and disadvantages of ether?
35 Discuss the physiological effects of an open thoracic cage which confront the anesthesiologist
36 What happens to the mediastinum when the thorax is opened?
37. What measures are instituted to correct these?
38 Why should positive pressure be applied intermittently?
39 What are the dangers when pressure exceeds twenty millimeters of water pressure?
40 Ir [illegible] culosis, how can spillage of infected m [illegible]
41. A [illegible] the lungs be done? Why?
42 What can be done to prevent postoperative pain?
43. Give the essential postoperative care of patients who have undergone thoracic surgery.

Chapter 5

ANESTHESIOLOGY—COMPLICATIONS DURING GENERAL ANESTHESIA

By Kenneth S. Weiss

Respiratory Complications.—During general anesthesia the respiratory are the most frequent complications encountered and if untreated will produce asphyxia and eventually death. Inadequate respiration may be the result of depression of the respiratory center, excessive amount of curare or curare-like drugs, or obstruction of the respiratory passages.

Respiratory complications arising from depression of the respiratory center are usually due to an overdose of the anesthetic agent, especially cyclopropane or Pentothal. They may also result from excessive pre-medication with narcotics, drugs or by obliterating the Hering-Breüer reflex. Narcotic drugs elevate the threshold of the respiratory center to carbon dioxide stimulation so that unless excessive amounts of carbon dioxide are allowed to accumulate or the amount of the drug is decreased the respiratory center will fail to respond to the usual stimuli and the patient becomes apneic. This phenomenon can also occur as a result of reduction of the carbon dioxide concentration of the blood due to hyperventilation. The Hering-Breuer reflex may be obliterated when the lungs are overinflated. In this event the afferent stimuli from the lungs, passing by way of the vagus nerve to the respiratory center, are cut off thus destroying the stimulus for physiological control of the respiratory movements.

The introduction of curare and curare-like drugs is one of the greatest advances in modern anesthesia. One objection to their use, is the impossibility of producing adequate relaxation without producing some degree of respiratory depression or even apnea. The treatment of overdosage is to give artificial respiration until the effects of the drug disappear. If the drug used is one of the ultra-short-acting curare-like drugs such as succinylcholine, the effects will usually wear off in a few minutes. If the drug used is one of the longer acting curare drugs, the respiratory complication is treated with one or two cubic centimeters of 1:2000 prostigmine mixed with $\frac{1}{150}$ of a grain of atropine and administered intravenously. Prostigmine is very effective in restoring postoperative respiratory depression when the patient has not as yet gained full control of his respiratory movements.

Respiratory obstruction, resulting from complete or partial block of the airway, is the most frequent complication of inhalation anesthesia. If untreated, the inevitable result is anoxia and carbon dioxide retention. The syndrome is characterized by inadequate tidal exchange, noisy respiration, and exaggerated movements of the thorax and diaphragm. If permitted to continue untreated, there will be an elevation of blood pressure, increase in the pulse rate, cardiac arrhythmia and cyanosis.

Obstruction caused by the tongue falling back against the posterior pharyngeal wall is usually due to relaxation of the muscles of the jaw and pharynx. To pre-

vent this, the tongue should be pulled away from the posterior pharyngeal wall and an oral pharyngeal airway inserted.

The obvious treatment for secretions and foreign bodies causing obstruction is their removal. Spasms of the adductor muscles of the vocal cords results in complete or partial obstruction. This is usually due to irritating anesthetic vapors, secretions, blood and foreign bodies in the pharynx, or it may result from trauma or sensitization of the vocal cords by the ultra-short acting barbiturates such as Pentothal or Surital. The treatment consists of removing the cause, deepening the plane of anesthesia and giving oxygen under pressure. If these measures fail, twenty to forty milligrams of succinylcholine given intravenously will relax the muscles and relieve the spasm.

Respiratory obstruction beyond the vocal cords due to a foreign body in the tracheobronchial tree is treated by bronchoscopic aspiration. Spasm of the tracheobronchial tree may be relieved by sympathomimetic drugs such as epinephrine or aminophylline.

Cardiovascular Complications.—Pathologic alterations of the cardiovascular system are frequently encountered during general anesthesia. Cardiac disturbances may vary from mild arrhythmia to complete asystole of the heart. Arrhythmia occurring in a patient with a normal heart is frequently encountered during chloroform or cyclopropane anesthesia and less frequently during ether or nitrous oxide anesthesia. Chloroform has a direct effect on the heart by increasing its irritability. Arrhythmia of ventricular origin occurs rather frequently with cyclopropane anesthesia due to increased carbon dioxide content in the blood. Treatment consists of increasing the patient's ventilation by manual compression of the breathing bag. Procaine amide which acts as a myocardial depressant also tends to prevent arrhythmia during cyclopropane anesthesia.

Cardiac arrhythmia of auricular origin is occasionally encountered during ether anesthesia. Sinus tachycardia is probably due to vagal inhibition or to stimulation of the sympathetic cardiac mechanism. Other types of cardiac abnormalities are rare and are usually due to an excessive amount of the anesthetic agent.

Nitrous oxide has no direct effect on the heart and alterations in cardiac rhythm are the result of hypoxia which may occur concurrently with the use of the drug.

Inadequate oxygenation, regardless of the type of anesthesia, usually causes an increase in the cardiac rate which is then followed by bradycardia. Severe loss of blood, shock, or trauma during surgery will be accompanied by tachycardia. Epinephrine and ephedrine will usually cause a transient increase in the heart rate.

Decrease in the heart rate may be the result of vagal stimulation and is frequently seen in thoracic or upper abdominal surgery. Should this occur, atropine sulphate, grains $\frac{1}{100}$ to $\frac{1}{75}$ given intravenously is very effective for blocking the vagus nerve.

Disturbance in bood pressure is frequently encountered during anesthesia. Elevation in blood pressure may be great enough to precipitate a cerebral vascular accident. Hypertension is most commonly the result of carbon dioxide retention due to either inadequate ventilation or faulty carbon dioxide absorption within the anesthesia apparatus. When the carbon dioxide retention is corrected the blood pressure will return to normal level. Inadequate oxygenation will also result in a temporary rise in blood pressure and will be followed by a drop in pressure if the condition is allowed to persist. Other causes of hypertension are thyrotoxicosis, increased intracranial pressure and overdosage of vasopressors.

Hypotension may be caused by any, or a combination, of the following abnormalities: (1) Blood loss; (2) surgical trauma; (3) perforated viscus, and (4) overwhelming toxemia. In all of these, an attempt should be made to treat the underlying causative factor producing the hypotension prior to surgery, if possible. During surgery, hypotension may result from undue traction of the mesentery or abdominal viscera. This can be treated by means of vasopressors. Hypotension will invariably occur following spinal anesthesia unless a vasopressor drug is administered prophylactically prior to the spinal anesthesia.

Cardiac arrest may occur at any time during an operative procedure under general anesthesia. Usually there is a preexisting period of hypoxia which manifests itself by cyanosis, hypotension, or cardiac arrhythmia. If the blood pressure cannot be obtained and there is no palpable pulse, either peripherally or over the great vessels, and there is an absence of bleeding at the operative site, cardiac arrest must be suspected. However, before a plan of action is instituted for cardiac arrest, severe circulatory collapse must be ruled out. This can be done by administering a small amount of a potent vasopressor such as neosynephrine. If the heart is still functioning, even though pulsations cannot be felt, neosynephrine will cause a rise in the blood pressure and may be the only treatment necessary. If this procedure fails, then a definite plan of action must be taken. Circulation and respiration must be maintained artificially until the heart and respiratory center can function normally. This can be accomplished only by manual systole of the heart by the surgeon and manual compression of the breathing bag by the anesthetist to maintain respiration.

The only method to insure adequate circulation is to approach the heart through the chest wall. A transverse incision is made in the fourth or fifth interspace extending from the left of the sternum laterally for about eight inches. The heart is grasped in the hand between the thumb and four fingers and squeezed in such a fashion that the blood is expelled in sufficient quantity to produce a palpable pulse. This is followed by a period of relaxation simulating diastole and the maneuver is again repeated. This should be done about 40 to 60 times per minute.

If the surgery is an intra-abdominal procedure the heart may be compressed through the intact diaphragm while the chest is being opened. This is only a temporary expedient as the heart can only be reached properly through the chest wall

At the same time, the lungs must be inflated with 100 per cent of oxygen by manual compression of the breathing bag at the rate of 15 to 20 per minute. A tight-fitting mask over the patient's face may suffice, however a cuffed endotracheal tube is preferred. The operating table should be placed in a 5- to 8-degree Trendelenburg position to aid the return of the blood flow to the heart and to increase cerebral circulation.

Cessation of cardiac activity may be either a ventricular standstill or a ventricular fibrillation. Since the treatment for the two conditions differs, it is important to differentiate the two. In ventricular standstill, the more common of the two, manual systole may be sufficient to start the heart beating spontaneously. As an adjuvant, 0.5 cc. of epinephrine mixed with 9.5 cc. of 1 per cent procaine may be injected into the right ventricle. The procaine is mixed with epinephrine to prevent the heart from fibrillating without affecting the action of epinephrine. Calcium chloride, 5 cc. in 10 per cent solution, is also very effective in stimulating myocardial contractions.

In ventricular fibrillation the heart presents the characteristic appearance of a

"writhing bag of worms." Although there is activity in the cardiac muscle, there is no cardiac output. The fibrillation must be stopped before the heart can again beat effectively. Ten cubic centimeters of one per cent solution of procaine injected into the right ventricle is very effective in reducing cardiac irritability and may stop the fibrillation. If this is inadequate an electric current may be passed through the heart to "defibrillate" the ventricles. Two electrodes are placed on either side of the heart and a current of one to one and one-half amperes, for 0.3 of a second, is passed through the heart. This is done by a special defibrillating apparatus which should be standard equipment on all operating floors. When the heart has stopped fibrillating it will be in asystole and manual systole is continued until the heart resumes its normal activity. When the heart functions under its own stimulus for a period of ten to fifteen minutes the chest wall is closed and a water seal drainage is inserted.

Gastro-intestinal Complications.—Emesis, and the danger from aspiration into the tracheobronchial tree, must be always guarded against during general anesthesia. Although retching and emesis do not occur as frequently today as a result of the utilization of barbiturate induction, adequate facilities must be made available to prevent serious complications. A patient who has eaten within six hours prior to surgery should not be given a general anesthesia. In an emergency case, the stomach must be emptied before the administration of the anesthetic.

Emesis during induction is less likely to occur when short-acting barbiturates rather than when inhalation agents are used. Emesis can also occur should the anesthesia become inadequate during the surgical procedure or at its termination when the patient passes from surgical to the lighter planes of anesthesia. Emesis during induction must be especially guarded against in patients with upper gastro-intestinal bleeding and intestinal obstruction. These cases should be intubated under topical anesthesia with a cuffed endotracheal tube while they are awake. The cuff is inflated to seal off the trachea and the patient can then be anesthetized without fear of aspiration of gastric contents.

Should emesis occur during general anesthesia, in spite of all the precautions, the head end of the table is lowered and the patient's head is turned to the side to prevent the gastric contents from entering the trachea. A suction apparatus must be available at all times to aspirate any contents from the posterior pharynx and trachea.

Convulsions.—Another complication that may occasionally occur during anesthesia is involuntary muscle contractions that may progress to true convulsions. This may be the result of hypoxia, excessive carbon dioxide accumulation or stimulation of the motor centers by such drugs as Vinethene.

Pre-existing diseases of the central nervous system may also predispose to convulsions. The treatment consists of providing adequate oxygenation and carbon dioxide elimination and reducing the concentration of the anesthetic agent.

QUESTIONNAIRE

1. Which are the most frequent complications after a general anesthesia?
2. Inadequate respiration is the result of what factors?
3. [illegible] complications?
4. [illegible]
5. [illegible]
6. What are the objections to the use of curare and curare-like drugs?

7. What is the treatment of overdosage with the above drugs?
8. What drug is used to restore postoperative respiratory depression?
9. What is the most frequent complication of inhalation anesthesia?
10. What is the syndrome of respiratory obstruction?
11. How is an obstruction caused by the tongue falling back against the posterior pharyngeal wall to be prevented?
12. What are the causes for spasm of the adductor muscles of the vocal cords? Treatment?
13. What drugs are used to treat spasms of the tracheobronchial tree?
14. What cardiac disturbances may occur during general anesthesia?
15. Which anesthetic agents frequently cause arrhythmia?
16. What is the effect of chloroform on the heart?
17. Which anesthetic agent is often followed by arrhythmias of ventricular origin?
18. What is the treatment for arrhythmia?
19. What is the occasional cause of arrhythmia of auricular origin?
20. What are the possible causes of sinus tachycardia?
21. Does nitrous oxide have any direct effect on the heart?
22. What is the effect of inadequate oxygenation?
23. What may cause tachycardia?
24. What is the effect of epinephrine and ephedrine on the heart rate?
25. What may cause a decrease in the heart rate?
26. What drug would you use to treat the above? Why?
27. What is the most frequent cause of hypertension during general anesthesia?
28. What are the causes of hypotension?
29. What are the signs and symptoms indicating possible cardiac arrest?
30. How can you differentiate between severe circulatory collapse and cardiac arrest?
31. What is the most urgent and specific requirement to restore cardiac function?
32. Where would you make the incision to expose the heart?
33. Describe the technique of manual compression of the heart.
34. How many times per minute would you compress the heart?
35. What is the role of the anesthetist during cardiac arrest?
36. How much oxygen should be used and how is it administered?
37. What should the position of the operating table be during this period? Why?
38. What is the difference between ventricular stand-still and ventricular fibrillation?
39. What is the treatment for ventricular stand-still?
40. What is the characteristic appearance of the heart during ventricular fibrillation?
41. Since fibrillation must be stopped before the heart can beat again, what drug is recommended for this purpose?
42. When should the electric current be used?
43. Describe the technique of using electric stimulation.
44. How long after the heart functions under its stimulation should the chest be closed?
45. Should a patient who has eaten six hours prior to surgery be given a general anesthesia? How about in the emergency case?
46. How would you treat a patient who has emesis during general anesthesia?
47. What is the cause of convulsions during anesthesia?
48. What is the treatment for the above?

"writhing bag of worms." Although there is activity in the cardiac muscle, there is no cardiac output. The fibrillation must be stopped before the heart can again beat effectively. Ten cubic centimeters of one per cent solution of procaine injected into the right ventricle is very effective in reducing cardiac irritability and may stop the fibrillation. If this is inadequate an electric current may be passed through the heart to "defibrillate" the ventricles. Two electrodes are placed on either side of the heart and a current of one to one and one-half amperes, for 0.3 of a second, is passed through the heart. This is done by a special defibrillating apparatus which should be standard equipment on all operating floors. When the heart has stopped fibrillating it will be in asystole and manual systole is continued until the heart resumes its normal activity. When the heart functions under its own stimulus for a period of ten to fifteen minutes the chest wall is closed and a water seal drainage is inserted.

Gastro-intestinal Complications. Emesis, and the danger from aspiration into the tracheobronchial tree, must be always guarded against during general anesthesia. Although retching and emesis do not occur as frequently today as a result of the utilization of barbiturate induction, adequate facilities must be made available to prevent serious complications. A patient who has eaten within six hours prior to surgery should not be given a general anesthesia. In an emergency case, the stomach must be emptied before the administration of the anesthetic.

Emesis during induction is less likely to occur when short-acting barbiturates rather than when inhalation agents are used. Emesis can also occur should the anesthesia become inadequate during the surgical procedure or at its termination when the patient passes from surgical to the lighter planes of anesthesia. Emesis during induction must be especially guarded against in patients with upper gastro-intestinal bleeding and intestinal obstruction. These cases should be intubated under topical anesthesia with a cuffed endotracheal tube while they are awake. The cuff is inflated to seal off the trachea and the patient can then be anesthetized without fear of aspiration of gastric contents.

Should emesis occur during general anesthesia, in spite of all the precautions, the head end of the table is lowered and the patient's head is turned to the side to prevent the gastric contents from entering the trachea. A suction apparatus must be available at all times to aspirate any contents from the posterior pharynx and trachea.

Convulsions.—Another complication that may occasionally occur during anesthesia is involuntary muscle contractions that may progress to true convulsions. This may be the result of hypoxia, excessive carbon dioxide accumulation or stimulation of the motor centers by such drugs as Vinethene.

Pre-existing diseases of the central nervous system may also predispose to convulsions. The treatment consists of providing adequate oxygenation and carbon dioxide elimination and reducing the concentration of the anesthetic agent.

QUESTIONNAIRE

1. Wh...
2.
3. ... general anesthesia?
4.
5.
6.

7. What is the treatment of overdosage with the above drugs?
8. What drug is used to relieve postoperative respiratory depression?
9. What is the most *frequent complication of inhalation anesthesia?*
10. What is the syndrome of respiratory obstruction?
11. How is an obstruction caused by the tongue falling back against the posterior pharyngeal wall to be prevented?
12. What are the causes for spasms of the adductor muscles of the vocal cords? Treatment?
13. What drugs are used *to treat spasms of the tracheobronchial tree?*
14. What cardiac disturbances may occur during general anesthesia?
15. Which anesthetic agents frequently cause arrhythmia?
16. What is the effect of chloroform on the heart?
17. Which anesthetic agent is often followed by arrhythmia of ventricular origin?
18. What is the treatment *for arrhythmia?*
19. What is the occasional cause of arrhythmia of auricular origin?
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Chapter 6

ANESTHESIOLOGY POSTOPERATIVE ANESTHETIC COMPLICATIONS

By Kenneth S. Weiss

PULMONARY COMPLICATIONS

The pulmonary are the most frequent postoperative complications encountered following general anesthesia. They are more apt to occur in upper rather than lower abdominal surgery. Patients with pulmonary disease, heavy smokers with profuse bronchial secretions, the aged and the markedly debilitated also have a higher incidence of pulmonary complications. Lastly, there is a correlation between the duration of the operative procedure and the depth of the anesthesia to the number of complications that may subsequently develop.

Atelectasis.—Atelectasis may be defined as partial or complete collapse of the lung due to the inability of the alveoli to expand completely. Any condition which will cause an increase in intrathoracic pressure, such as pneumothorax or pleural effusion, may prevent complete distention of the alveoli. Mucous plugs in the bronchi and bronchioles, by obstructing the air passages, prevent air from entering the alveoli, thus making it impossible for the affected portion of the lung to expand. Anything that will prevent the patient from expanding his lungs, such as tight abdominal and chest binders, may cause atelectasis.

The symptoms of atelectasis usually occur about forty-eight hours following surgery but in some cases may not occur until the tenth postoperative day, or later. Depending upon the extent of the lung involvement, the history is one of sudden onset of pain in the lower chest, dyspnea, cyanosis, increased respiratory rate and tachycardia The most important physical sign is asymmetrical movement of the chest wall; diminished or absent movement on the affected side and excessive on the normal side. The diagnosis is usually verified by *x*-ray.

Prophylactic measures to prevent atelectasis should be instituted in every postoperative case following a general anesthetic. Routine use of narcotics, especially large doses, must be avoided because they depress the cough reflex and ciliary activity thus permitting secretions to accumulate in the tracheobronchial tree. Early ambulation and breathing exercises should be instituted to encourage normal expansion of the lungs and to prevent stasis. Carbon dioxide (100%) is a very potent respiratory stimulant which causes the patient to breathe deeply even though he has *no* desire to do so. It is administered every few hours during the first or second postoperative day. It is supplied until the patient shows signs of hyperventilation, usually occurring after five or six good deep breaths. In the meantime the patient should be encouraged to cough. Many patients are afraid to cough, for fear of "breaking the incision." It is imperative to dispell this fear from their minds. Early ambulation is also essential, this is especially valuable during the first 48 hours following surgery. Aspiration of the tracheobronchial

thrombus. The prophylactic treatment consists of early ambulation, passive movements and exercises. When thrombosis has developed, the patient should be at bed rest with the extremities elevated. Anticoagulants are given in sufficient quantities to increase the coagulation time to twice that of the normal time. Lumbar paravertebral block may be used to block the sympathetic ganglia to abolish the reflex vasospasm; it also relieves pain and diminishes edema. Proximal vein ligation must be considered if the thrombus has extended into the thigh or if embolism occurs.

Phlebothrombosis is an aseptic clotting of the blood in a vein which is loosely attached to the wall of the vein. For this reason walking or any manipulation of the involved area may result in detachment of the clot with a resulting embolism. Phlebothrombosis may be asymptomatic, although examination of the involved extremity will reveal fullness and tenderness on palpation of the calf. Homan sign is usually positive. The treatment is early ligation of the femoral vein if th deep veins of the leg are involved

Complications Following Spinal Anesthesia. - Complications occurring postopera tively following spinal anesthesia are similar to those following general anesthesi However, there are certain complications which are specific for spinal anesthesia.

Headache is probably one of the most common complaints following spin anesthesia. It is the result of a disturbance in the hemodynamics of the spin fluid as a result of escape of spinal fluid following the puncture in the dura. Th headache is characterized by pain in the occipital region when the patient is in th upright position and relieved when lying down.

Prophylactic measures employed to reduce the post spinal headache are: (1 The use of a small-gauge needle to avoid a large puncture wound in the dur (2) the direction of the level of the spinal needle should be along the fibers of th dura to avoid cutting the fibers directly across, and (3) provide adequate hydratio to restore the normal volume of spinal fluid.

The therapeutic measures which may be utilized for treatment of headache are the use of aspirin or codeine for pain, and nicotinic acid to dilate the cerebra blood vessels. The patient should be kept in the most comfortable position Abdominal compression to elevate the cerebral spinal fluid pressure may be tried Epidural or caudal instillation of 40 cc. of saline has also been used to elevate th spinal fluid pressure and thus relieve the headache.

Meningismus or aseptic meningitis is another complication that occasionall follows spinal anesthesia. The symptoms occur on the third or fourth postoperative day. They are characterized by severe headache, rigidity of the neck, positive Kernig's sign, photophobia and occasional vomiting. Spinal tap reveals a high cell count of polymorphonuclear leucocytes and increased intradural pressure. Symptoms are usually transient, but may last for several weeks

Septic meningitis usually occurs within forty-eight hours after the operation. It is characterized by severe headache, elevated temperature, paresthesia and unconsciousness. Spinal fluid culture reveals a high white count and a positive culture for bacteria. Treatment consists of intense antibiotic therapy.

Other complications that may occur are arachnoiditis, myelitis and cauda equina syndrome. These are, as a rule, the result of neurotoxic effect of the anesthetic agent on the spinal cord and meninges. To avoid these complications, the least toxic drugs and with the lowest concentration should be used.

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$_{27}Co^{60}$ Radioactive Co	$\rightarrow$	$_{28}Ni^{60}$ Stable	+	B^- 0 31 mev	+	γ 1.33 mev	+	γ 1.17 mev			
$_{6}C^{14}$ Radioactive Carbon	$\rightarrow$	$_{7}N^{14}$ Stable	+	B^- 0.155 mev							
$_{11}Na^{24}$ Radioactive Sodium	$\rightarrow$	$_{12}Mg^{24}$ Stable	+	γ 1.37 mev	+	γ 2.75 mev	+	γ 4.13 mev	+	B^- 1.39 mev	+ B^- 4.17 mev
$_{88}Radium^{226}$ Unstable	$\rightarrow$	$_{86}Radon^{222}$ Unstable	+	$_{2}He^{4}$ Alpha particle							

The radioactive isotopes in their progress toward stability disintegrate by Alpha, Beta, Positron and Gamma emissions. An Alpha particle is the nucleus of a Helium atom, $_2He^4$. The Beta particle, (B^-), is an electron and has a mass of 0.00055 and unit negative charge. The Positron (P) has the mass of an electron but unit charge of opposite sign. A Gamma ray is electromagnetic radiation originating in the nucleus and is a similar type of radiation to that obtained from x-ray machines. It is these rays and particles that are emitted during atomic disintegration of radioactive materials that are of diagnostic and therapeutic importance. These rays and particles when used in therapy produce physical and chemical damage to cellular nuclear structure producing changes leading to cessation of mitosis and cell death, as well as differentiation to a more mature and therefore less malignant type of cell.

Alpha particles have a range (from 2.7 cm. to 8.6 cm.) in air, however since these particles can be stopped by a single sheet of paper, they are as yet of little importance in therapy. Beta rays generally have path lengths ranging from 1 mm. or less to 1 cm. in tissue. All Beta rays are stopped, by 2 mm. brass, 1 mm. of lead or silver and 0.5 mm. platinum. The lesser energy Beta rays are, of course, stopped by lesser quantities of heavy metal filtration. Beta rays or particles are of therapeutic value when thin layers of tissue are treated or when the Beta emitting isotope selectively concentrates in a specific tissue, or when the isotope can be injected and spread diffusely throughout a tumor bearing area. Beta rays likewise are of little diagnostic value when knowledge of their localization within the body is desired by external counting. Gamma rays generally have wave lengths shorter than those of medium voltage x-rays, and consequently greater penetrating powers and may have a path of many feet in tissue. A Gamma ray-emitting isotope within the body can be detected by external counting and therefore its localization within the body determined.

Nuclear disintegration is not a haphazard procedure but one which continues at a definite rate for the isotope involved and which follows certain mathematical laws. Any quantity of an element contains 6×10^{23} atoms per gram molecular weight. Every instant a certain proportion of atoms of radioactive material disintegrate making the original number of atoms present in the original sample that much smaller. With very few exceptions every atom of a given substance disintegrates in the same way. $_{15}P^{32}$, for example always disintegrates with the emission of a Beta particle of 1.70 million electron volts and the production of an atom of $_{16}S^{32}$. Every atom of Radium $_{82}B^{214}$ always disintegrates with the emission of B^- ray producing an atom of Radium $_{83}C^{214}$. Likewise each radioactive substance has its own particular rate of disintegration; that is, a particular percentage of the atoms in a given sample disintegrates per unit period of time. As few as 1 per cent of the atoms of a specific radioisotope may disintegrate per unit period of time while nearly all atoms of another radioisotope may have disintegrated in the same

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charge within the counter which is easily measurable and generally recorded by an electronic scaler or count rate meter. This type of detector will count ionizing particles which pass through the chamber walls and enter the sensitive volume. Gamma and x-rays are counted when a secondary electron is emitted from the chamber walls or from the gas itself.

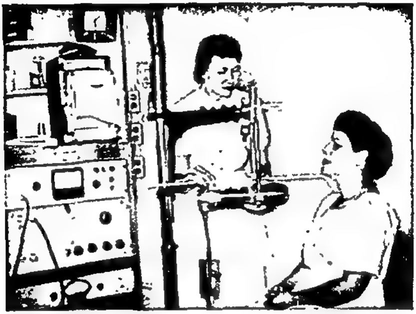

FIG. 7.—Scintillation counter with recording equipment in background.

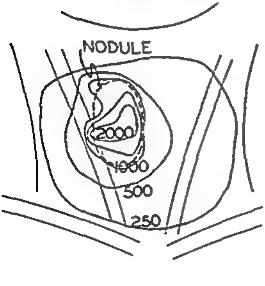

FIG 8

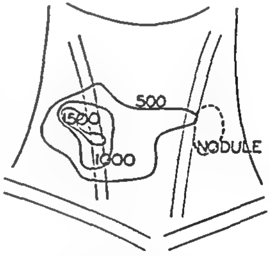

FIG. 9.

FIG. 8.—Diagram of scan of functioning nodule with a highly collimated scintillation counter.

FIG. 9.—Diagram of scan of nonfunctioning nodule with a highly collimated scintillation counter.

The scintillation counter (Figs. 6 and 7) is based on the phenomenon wherein energy lost by ionizing particles in certain crystals, is absorbed and re-radiated as light by the crystal. The amount of light given off is proportional to the amount of energy lost in each incident. The light is then collected onto the sensitive surface of a photomultiplier tube (ultrasensitive photo-cell). Each scintillation of light is changed into an electrical impulse which after being amplified can be recorded or

Chapter 8

THERAPEUTIC USES OF RADIOISOTOPES IN SURGERY AND RADIATION THERAPY

By Joseph P. Concannon

It is now almost fifteen years since the first clinical use of artificial radioactive isotopes in medicine. The clinical value of many isotopes have now been well-proven and some patients will be inadequately examined and treated if we do not make appropriate use of the various radioisotope methods. Isotopes have a diagnostic as well as a therapeutic usefulness in medicine and surgery. Furthermore tracer techniques with radioactive materials have been widely applied not only by clinicians but by workers in Biochemistry, Physiology, Pharmacology and other allied fields to learn more concerning the metabolism of ingested elements. It is not desired to becloud the surgical uses of radioisotopes in a textbook of surgery by an attempt to discuss the myriad uses to which they have been put. Rather an attempt will be made to discuss the use of those isotopes which have proven their value in surgical practice.

Radiation therapists and surgeons have long used radioactive materials in the treatment of patients with malignant disease. Radioisotopes used in therapy should be of interest to the embryonic as well as to the finished surgeon since they may be used for the primary treatment of malignant disease, as well as adjunctive therapy, preoperatively or postoperatively. Isotopes may be prepared in the form of needles, capsules, seeds, pellets, wires, colloidal suspensions and used as substitutes for radium and radon.

One of the most important uses to which radioactive isotopes have been put has been the development of relatively inexpensive teletherapy units which will do the work of the more expensive and cumbersome electrical supervoltage *x*-ray machines. Radioactive Cobalt, because of its availability, small physical dimensions, high energy and high Gamma-ray output, as well as relative cheapness has been widely used for teletherapy. The Cobalt "Bomb" is a large quantity of radioactive Cobalt, housed in a large container which is usually so constructed that quantities of lead or tungsten prevents the escape of the Gamma-ray beam in every direction except by way of a channel, the aperture to which may be opened or closed by remote control. The Gamma-ray beam emerging from the unit has similar physical characteristics to a beam of supervoltage *x*-rays. The quantity of radiation measured at a specific depth within a patient, expressed as a percentage of skin dose, is almost the same as that quantity of radiation delivered at the same depth by a 2- to 3-million peak voltage *x*-ray machine. Likewise, the biological effect on the tumor so treated will be similar whether a 3-million volt *x*-ray machine, or a $Cobalt^{60}$ beam unit both with an 80 cm. source to the skin distance.

There are many advantages of $Cobalt^{60}$ teletherapy and other supervoltage units over *x*-ray machines in the range of 200 to 400 kilovolts. The ratio of depth dose to surface dose is much greater than that of the lower energy *x*-ray machines.

the Cobalt^{60} source and its half-life is thirty-three years. The mean Gamma energy of radio Cesium, however, is approximately only one-half that of the Cobalt^{60}.

Radioisotopes have been used as a substitute for radium and radon in tumor therapy. Cobalt^{60} again was one of the first isotopes used as a substitute for radium in interstitial and intracavitary radiation therapy. The Gamma rays given off by Cobalt^{60} are essentially monochromatic, 1.1 and 1.3 million electron volts. The 0.31 million electron volt (max) Beta ray is removed by a metal sheath. When the radiation emitted from a mc. of Cobalt^{60} is measured it is found to be 13.6 roentgens per hour per millicurie at one centimeter, as compared with 8.4 roentgens per hour per milligram of radium. 0.62 millicurie of Cobalt therefore is equivalent to 1 milligram of radium. It is possible to use the conventional radium dosimetry tables and graphs in the calculation of radiation dosages to be given, when this correction is made. Cobalt^{60} sources are solid metal and have a more uniform field along the radiation source than radium sources. Furthermore, many of the older radium

FIG 11.—Radium needle implant of a malignant lesion of the tongue. Cobalt^{60} needles could be used for the same purpose.

needles and tubes have a remarkedly poor distribution of the radium salt; the salt having concentrated at one end of the needle or tube over a period of years. Cobalt^{60} is relatively inexpensive as compared with radium although the fact that it will decay to half its activity in 5.3 years makes necessary 6 per cent corrections at 6-month intervals. The dimensions of the Cobalt^{60} needles and tubes are essentially the same as those of conventional radium needles and tubes.

Cobalt^{60} needles have been used in implantation of tongue, anal, floor of mouth carcinomas as well as those carcinoma in other sites where radium needles have been previously used (Fig. 11). A disadvantage in the use of Cobalt^{60} for interstitial use is that metallic Cobalt is absorbed from the tissues and deposits in other parts of the body. There is some danger to its use interstitially even if sheathed because of the risk of breakage of the sheath material and leak of Cobalt from the ends Cobalt has been made in the form of tubes for intracavitary and mold radiation therapy and have been used in treatment of carcinoma of the cervix, body uteri, vagina, bladder and maxillary antrum. A recent advance has been the production of Cobalt^{60} in minute pellets. These pellets have been placed in nylon tubing and the tubing containing active Cobalt pellets then threaded through

in mold therapy for neoplasm of the buccal mucosa, floor of mouth, hard palate and lip. Lederman and Trott have sewn plastic tubing in rings containing Tantalum[182] onto the posterior portion of the globe of the eye for the treatment of retinoblastoma, replacing radium applicators and radon seeds that had been previously used. Likewise, they have used radioactive Tantalum mounted on fenestrated plastic shell for the treatment of epibulbar malignant melanomata, where treatment of the entire conjunctival sac was felt to be indicated. Wallace pioneered the interstitial use of Tantalum[182] wire as the interstitial radioactive source for treatment of small bladder tumors which have not extended through the muscularis of the bladder (Fig. 13). Ellis has introduced Tantalum[182] wire through a cannula into perirectal tissues for the treatment of inoperable carcinoma of the rectum.

FIG. 13.—Tantalum[182] implant for carcinoma of the bladder. (Courtesy D. Wallace, The Royal Marsden Hospital.)

Short lived materials are advantageous as interstitial sources in sites where it is not desired to remove the source at the end of the period. Radon seeds, with a half-life of 3.825 days have long been used in radiotherapeutic practice for permanent implants. Pendergrass has recently pointed out the lack of uniformity of radon seeds procured from commercial companies in the United States. When millicurie seeds were ordered later assay proved that these seeds contained from no radon at all to several millicuries per seed. This erratic quantity of radon could lead from failure to cure to necrosis of tissues as well as the so-called supralethal effect, viable cancer tissue associated with necrosis. Furthermore, it is difficult to construct a really satisfactory introducer for the seeds. Although an automatic ejecting device had been developed several years ago at the London Hospital it was

of Chicago inserted Yttrium^{90}, a pure Beta emitter into the sphenoid for the same purpose (Fig. 16). Attempts have been made to do volume implants with Gold^{198} grains at the time of surgery for unresectable bronchogenic carcinoma or for residual mediastinal disease.

Colloidal radiogold has been used in the infiltration of malignant tumors in areas where radium or radon seeds had been used previously. The colloidal Gold^{198} is not screened by Platinum as are the gold grains and therefore it is the 0.96 million electron volt Beta ray that is of importance in this form of therapy rather than the 0.41 million electron volt Gamma ray. Radiogold tends to remain localized

FIG. 15.—Gold^{198} grain implant for carcinoma of the bladder. (Courtesy D. Wallace, The Royal Marsden Hospital.)

at the site of injection and for this reason has been used in infiltration of tumor masses in supraclavicular, axillary, and other accessible locations. It is difficult, however, to achieve an even distribution of the colloid because of the resistance encountered in injecting the fluid into malignant tumors. Histological examination of tumor masses following Gold^{198} colloid therapy showed no evidence of even dispersion of the active material within the cancer. On the other hand the Gold^{198} colloid tends to leak out of friable and necrotic tumors.

Experimental studies have been done with radiogold colloid to determine its effect on the meningeal system with a view to its use for radiation therapy for such meningeal spreading tumors as medulloblastoma and ependymoma grade IV

cators. This occasionally has resulted in disastrous high dose effects as well as a lower cure rate. In an attempt to increase the dosage at the lateral parametria the use of colloidal Gold^{198} injected parametrically was studied by members of the Mallinkrodt Institute and the Department of Obstetrics and Gynecology of Washington University Medical School.

Approximately 70 mc. of Gold^{198} colloid was injected into each parametria through a 22-gauge needle inserted through the fornix superiorly and laterally. Following injection of radiogold colloid, particles of Gold^{198} were phagocytosed and carried to regional lymph nodes where radiation effects were noted due to phagocytosed Gold^{198}. There were, however, also other contributions to dosage in this area. It was estimated that there was an average local dose of 10,600 roentgens equivalent physical dose to Beta radiation of the Gold^{198}; 2,250 roentgens due to Gamma radiation of Gold^{198} plus a dosage of 3,350 roentgens due to intracavitary radium source. This dosage is far in excess of what one can obtain with radium and external radiation without disaster. This study is an important one and should be followed carefully. However, it falls within the realm of experimental radiation therapy and until, and if, the 5-year survivals obtained with this technique exceed those obtained with radium and external radiation it should not be adopted other than in research institutions.

The palliative use of Gold^{198} colloid instilled into pleural or peritoneal cavities for carcinomatosis with serous effusion was first described by Müller. His findings have since been confirmed by numerous investigators. There has been reduction of fluid accumulation in more than 50 per cent of patients with pleural and peritoneal effusions, excluding those patients who died in the first month following instillation of radiogold. Some patients may, however, re-accumulate fluid until several weeks have elapsed from the time of treatment. Although palliation has been good there has been no increase in the survival time of the patients treated. Most of the radiogold injected into the body cavities is fixed on or near the surface of the lining of the cavities partly in phagocytes and partly absorbed onto the serous surfaces. The gold colloid remains in pleural and peritoneal cavities, except a small amount which goes to local and regional nodes, with a very small fraction gaining access to the blood stream. The amount of activity remaining in free pleural or peritoneal fluid after one week is usually less than 10 per cent, and thoracentesis or paracentesis may be done if necessary at that time.

The cause of fluid reduction is not precisely known, and several mechanisms have been suggested: A radiation lethal effect on small malignant seedling on several surfaces, a lethal effect on free cancer cells in fluid, or finally radiation effects and fibrosis of mesothelium, tumor surfaces and small blood vessels in the serosa.

The adverse effects of treatment are usually negligible. There is a mild and transitory radiation sickness associated with prompt lymphopenia and later granulocytopenia and thrombocyopenia Only a few cases of hypoplasia of the bone marrow have been reported with use of radiogold and those occurred in patients who were in the terminal phases of their malignant disease. The greatest hazard to the patient is when the administered Gold^{198} colloid becomes loculated in the small segment of the pleural or peritoneal cavities. These small areas then may receive a remarkably high local dose of radiation and bowel necrosis and perforation may occur. To minimize those undesirable complications it is wise to scan the abdomen or chest following the instillation of radiogold colloid to determine

CHAPTER 9

SUTURE AND LIGATURE MATERIAL

BY A. V. PARTIPILO

"All the affairs of men hang by a slender thread; and sudden change brings to ruin what was once strong."—OVID.

WHEN blood vessels greater than the dimensions of capillaries are divided, it is necessary to tie the divided ends. The material employed for this purpose is called a ligature. The material used to sew the tissues together until healing of the wound takes place is called a suture.

Ligatures have been used for hemostatic purposes for centuries before the Christian Era. Celsus refers in his works to the ligature, and credits its discovery to a physician of the Alexandrian school. Galen frequently mentions the ligature and gives particular directions to apply it to the proximal end of a divided vessel. Until the time of Paré ligatures were used as a last resort, whereas styptics and the actual cautery were generally relied upon to arrest hemorrhage. Through the influence of Paré the ligature eventually became popular, however, the dangers of using contaminated ligatures were not fully appreciated until Lister discovered the cause of sepsis and introduced "antiseptic catgut." Nevertheless, wound infections and complications continued to plague the surgeon until Meleney, Clock, and others proved that chemical sterilization is unreliable and that heat is the only effective method of sterilizing catgut. At present, catgut is heat-sterilized and must conform to the standards outlined in the U. S. Pharmacopeia XIV.

THE PERFECT LIGATURE

Notwithstanding the great amount of clinical and experimental research work, there is still a wide difference of opinion as to the choice of ligature and suture material. Every surgeon has his preference; however, there are not many who prefer any one kind of suture to the exclusion of others. The advocates of the silk technique condemn catgut as an impossible suture material; those who employ catgut find fault with non-absorbable sutures; and those who use metallic wire find this material an incomparable suture and object to any other type of material. In the meantime, the pendulum swings back and forth in periodic cycles and this will continue until some one comes along and introduces the "perfect ligature." A perfect ligature or suture must conform to the following requirements:

1. Must be free from bacteria.
2. The material must not act as a culture media for the growth of bacteria which may enter the wound.
3. The material should withstand sterilization without destroying its efficiency.
4. It should hold the edges of the wound in apposition until union of sufficient strength has been established, regardless of the length of the "lag period."

NON-ABSORBABLE MATERIALS

A. **Metallic Origin.** 1. Gold is one of the oldest materials used in surgery. It is non-cytotoxic and produces minimum tissue reaction, but because of its cost it is seldom used.

2. Silver has had extensive use in bone surgery, but it is also being replaced by newer metals. In 1911 Harvey Cushing introduced the silver clip to produce hemostasis in neurosurgery. This is probably the most important use of silver as a ligature. Pudenz, in 1942, found that silver clips provoked intense inflammatory reaction and dense connective tissue response. As a result of his experimental studies, he believed that the silver clip should be replaced by tantalum.

3. Copper and bronze have likewise been replaced by the newer metals. Copper is definitely cytotoxic and should not be used.

4. Vitallium, vanadium, ticonium, stainless steel, and tantalum have been introduced recently. Vitallium is an alloy containing 65 per cent cobalt, 30 per cent chromium, and 5 per cent molybdenum. It is used in bone surgery in the form of screws, nails and plates. It cannot be drawn into wire nor rolled. Vanadium steel is seldom employed because it causes intense local reaction. Tantalum is a bluish-white basic element having many of the chemical properties of glass and physical characteristics of steel. It is three times as heavy as iron, and its tensile strength is comparable to mild steel. It can be drawn into wire as fine as human hair or rolled into fine thin foil. Tantalum wire thread, having a diameter of 0.003 inches (B. & G. Gauge 40) has the tensile strength of number 6-0 catgut. It is non-cytotoxic, non-corrosive, and inert to any chemical that may be produced in the body. When placed in a wound there is an almost complete absence of tissue reaction. It can be used with safety as a buried suture and for suturing the skin. Table 3 gives the size number, diameter, and tensile strength of tantalum.

Stainless steel was introduced in 1934 by Babcock for use as a suture and ligature. This material is stainless, has marked tensile strength, and when buried in the tissue does not tarnish, discolor, nor irritate the tissues. It is used in place of silver and bronze in bone work. It may be substituted for dermal, silkworm-gut, silk,

and horsehair as an approximating skin suture. It is also recommended as a buried suture in the repair of tendons, in herniorrhaphies, and in gastrointestinal anastomoses. For delicate approximation and for ligating purposes, Babcock advises 36 or 35 B. & G. gauge; this is about the size of fine horsehair having the tensile strength of two and one-half pounds. For supporting suture, use 30 gauge with a tensile strength of 5 pounds. The larger sizes are employed in bone surgery.

5. Michels' metallic clips are preferred by many surgeons for approximating the skin edges. They are quickly and easily applied, and produce an approximation with varying degree of eversion. They can be removed on the third or fifth day, and leave a small scar. In an abdominal operation, after the clips are removed, adhesive strips are used for support. They are especially useful in approximating the skin edges after thyroidectomy. A fine scar results, and no marks are visible if the clips are removed on the second or third day.

TABLE 3.—SIZE, DIAMETER, AND TENSILE STRENGTH OF TANTALUM

B & S gauge	Size	Diameter inches	Breaking strength straight pull in pounds
40	000000	0 003	[illegible]
36	00000	0 005	1 70
33	0000	0 007	[illegible] 50
30	000	0 010	4 60
26	0	0 015	10 40
24	2	0 020	23 10

Metallic metals in orthopedic surgery must meet the standards of passivity or freedom from electro-activity in the body, which have been established by the Fracture Committee of the American College of Surgeons. The question as to the choice of metals and their suitability in surgery can be briefly summarized by quoting Venable, *et al.*, who state that, "Only three materials generally available seem to possess sufficient passivity to be used in the body: '18-8-SMO' stainless steel, which is relatively inert and can be adapted for small appliances such as wire or screws; tantalum, which is inert and will be useful when production problems are solved; and vitallium, which is inert, has been used widely, and has been found to be perfectly safe for any type of appliance, except wire, which requires malleability strength and the essential biochemical resistance."

B. **Animal Origin.**—1. Silkworm gut is made from the silk producing glands of the silkworm. It commends itself because it is non-capillary, hence does not absorb fluids. It is easily sterilized by boiling, is non-irritating to the tissues, and is non-absorbable. This material is not used as a buried suture. It is especially useful as a tension suture. Its stiffness may be overcome by soaking it in sterile water for a short time.

2. Horsehair is obtained from the tail of the horse. For fine plastic work on the skin, when a small scar is desirable, horsehair makes a fine suture. Its tensile strength is not very great, and for this reason it should not be used when tension on the suture line is anticipated. The chief objection to horsehair is its unreliability in its size and length. It also has the tendency to break while suturing, or when tying a knot. At present horsehair is seldom employed.

3. Dermal is made from carefully selected silk fibers, and is finished by a special process. It is supplied in various sizes, designated according to standard catgut gauges. Size 4-0 is recommended as a fine suture in plastic surgery. Sizes 000

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replaced by silk and cotton. Pagenstecher linen is made by impregnating linen thread with celluloid to render it more pliable. It has little capillary, hence it does not absorb fluids. Linen thread is very flexible and does not become tangled as easily as silk. Pagenstecher linen is of special value as the outer row of suture in an intestinal anastomosis. It is readily sterilized by boiling; repeated boiling does not impair its tensile strength. Size for size, it is comparable to silk in its tensile strength.

2. Cotton is a cellulose material which is spun into thread from fine fibers. Ordinary and mercerized spool cotton is made from the long fiber of Sea-island or Egyptian cotton. Ordinary cotton is of six twisted cords, while the mercerized is three cords. Mercerized cotton, made by treating with alkalies, is stronger than ordinary cotton. Since the original reports of Meade and Ochsner, cotton has found an ever widening use as a suture material. The observance of a careful technique is just as important with this material as with silk.

Table 5 gives the size, diameter, and tensile strength of cotton as standardized in U. S. P. XIV.

TABLE 5.—SIZE, DIAMETER, AND TENSILE STRENGTH OF COTTON

Size	Diameter in inches		Minimum tensile strength in pounds on straight pull
	Minimum	Maximum	
0000000	0.001	0.002	0.25
000000	0.002	0.004	0.50
00000	0.004	0.006	1.00
0000	0.006	0.008	2.00
000	0.008	0.010	3.00
00	0.010	0.013	5.00
0	0.013	0.016	7.00
1	0.016	0.019	10.00
2	0.019	0.022	13.00
3	0.022	0.025	16.00
4	0.025	0.028	20.00
5	0.028	0.032	25.00
6	0.032	0.036	30.00
7	0.036	0.040	35.00

The surgeon should determine before operating the size and tensile strength of the cotton suture furnished him, otherwise he is apt to use an altogether improper size. In order that the surgeon may employ the greatest possible strength of the suture, Localio *et al.*, suggests that cotton be sterilized by boiling from ten to twenty minutes, and used while wet. He suggests the following size cotton for the various tissues:

1. Ligating small blood vessels; Handcraft 000.
2. Ligating large blood vessels; Handcraft 00 or 0.
3. For suturing fascia and peritoneum; Handcraft A 40.
4. Intestinal anastomosis; Handcraft A 40.

Cotton should not be used as a continuous suture. Cotton sutures of tensile strength equal to the holding power of the tissue to which it is applied, may be used providing the number of interrupted sutures are sufficient in number. Shambaugh emphasizes the importance of placing interrupted stitches close together if accurate approximation and strength is desired. He states that, "The strength of the suture line does not increase in mathematical proportion with an increase in the number of stitches, probably because the strain is not evenly distributed,

been used. It has not been used extensively. In 1946 Chandy made a study of the fate of preserved heterogeneous grafts of fascia when transplanted into living tissues. His observations are interesting and should be studied by the surgeon who wishes to use ox fascia as a suture. His conclusions are:

(*a*) The preserved ox fascia can be identified after four years of implantation in human tissue.

(*b*) There is a very slow change in the character and nature of the preserved fibrous tissue cells.

(*c*) There is an abundant growth of capillaries and fibroblasts from the periphery into the fascial graft and complete absorption of the preserved fascial cells at the periphery.

(*d*) The absorption of the cellular detritus of the preserved fascia is noted.

(*e*) The fibers of the preserved fascia become rearranged into bundles as more fibroblastic and capillary ingrowth takes place.

(*f*) There is relatively little leucocytic infiltration at the periphery of the graft and between the bundles.

(*g*) There is no marked foreign body reaction and giant cells are absent.

Preserved ox fascia is not recommended in the ordinary type of hernia. It has a place in the extensive incisional hernia where there is great loss of fascia.

C. **Surgical Gut.**—Catgut is made from the submucous coat of the intestine of the sheep. It consists mainly of connective tissue with a few elastic fibers. Catgut is now supplied plain Type A, or treated with certain chemicals to render its absorption slower, and as boilable or non-boilable. The latter terms refer to the methods of sterilizing the exterior of the tube. Boilable catgut is dehydrated and supplied in a tube containing lysol or toluol. It is stiff, brittle, and difficult to handle. The non-boilable variety is not dehydrated and is supplied in tubes containing alcohol. The tube is sterilized by a chemical process and never by boiling.

Sizes and Tensile Strength.—Catgut is manufactured in sizes ranging from number 7-0 to number 7. These numbers refer to the diameter, and the tensile strength of catgut which must conform to the standards indicated in the U. S. Pharmacopeia XIV (see Table 6).

Absorbability of Catgut.—The one specific advantage of catgut is its absorbability. Plain catgut is absorbed or loses its tensile strength in a few days. Therefore, it is used only when rapid healing is expected, as when suturing serous and mucous surfaces, and for ligating small blood vessels. In order to decrease the rate of absorption, catgut has been treated with a variety of chemicals such as, chromic acid, iodine, tannin, silver, formalin, mercury, and others. At present chromic acid is universally used. The degree of chromitization was formerly designated as 10, 20, 30, or 40 day catgut, signifying the rate of absorbability. The new nomenclature is as follows: Type B, mild treatment, Type C, medium treatment, and Type D, prolonged treatment. According to Jenkins Type B retains its tensile strength for 5 to 10 days; Type C 10 to 15 days; and Type D 15 to 25 days. The length of time for complete absorption varies from 14 to 60 days depending upon the type. It must be emphasized that these are only relative terms, since they are based upon the rate of disintegration in the thigh muscles of test animals. In the human other factors must be taken into consideration. It is generally known that chromic catgut is absorbed much more quickly in the presence of infection. Since air-borne bacteria can hardly be eliminated from a wound, it is imperative that the technique employed does not enhance their growth. Trauma, hematomas,

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is nil, whereas the holding strength of the catgut is at its greatest. At the conclusion of healing the ligature has lost its strength or has disappeared entirely. Accurate measurement of these two factors should give us an indication as to the size and degree of chromitization of catgut to employ for different tissues.

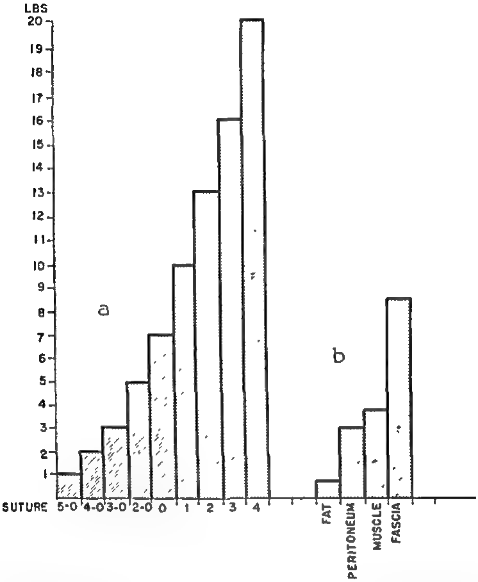

FIG. 19.—*A*, Breaking strength of catgut in pounds (vertical numbers). *B*, Holding strength of a single stitch of number 0 catgut in fat, peritoneum, muscle, and fascia at the time of insertion.

CATGUT TECHNIQUE

The following practical conclusions are drawn as to the breaking strength of catgut as compared with the tearing strength of different tissues (*see* Fig. 19):

(*a*) *Fat Tissue.*—From a practical point of view fat has no holding power, since any suture will tear through at less than one pound regardless of the size of catgut used. Hence, the smallest size of catgut obtainable should be used for coapting fatty tissue. As a matter of common knowledge, catgut is poorly tolerated in subcutaneous fatty tissue, and does not add to the strength of the wound. If it is necessary to obliterate the "dead space," suture the subcutaneous fascia found at

the lower level of the fatty layer with interrupted plain catgut number 5-0, combined with tension sutures.

(b) *Peritoneum.*—The holding strength of a single stitch in the peritoneum, while higher than fat, still remains below the strength of the finest catgut. The holding power of the small bowel is less than one pound so that chromic catgut number 5-0 as suggested by Bower is of sufficient strength. He also suggests this size for suturing the dura, tendons, nerves and for surgery of the biliary tract. Chromic catgut number 5-0 produces minimal cellular reaction; insures apposition of mucous and serous surfaces because twice as many stitches can be inserted within a given space, thereby providing a suture line of great tensile strength.

(c) *Muscle.*—The stitch tears out at a higher level than fat or peritoneum, but still below the breaking strength of 3-0 catgut. Muscle tolerates any kind of suture material poorly. If its sheath is carefully sutured there is no need for suturing the muscle itself. To obliterate dead space in a muscle, number 3-0 or smaller plain catgut may be applied loosely.

(d) *Fascia.*—Only in fascia and similarly condensed connective tissue layers is the holding power of the tissues greater than the tensile strength of number 3-0 catgut. Number 0 type C (chromic) catgut having a minimum tensile strength of seven pounds has more than the required strength for suturing the fascia. Larger sizes do not add to the strength of the wound and they may weaken it by causing an excessive tissue reaction.

(e) For ligating small bleeding points, plain catgut 4-0 is of sufficient strength. For ligating large blood vessels, chromic catgut 0 should be used because the end of the bleeding vessels should be held by a suture until healing has occurred by fibroplasia.

SUTURE TECHNIQUE

There is a great deal of controversy as to the choice of suture material to employ in surgery. Until such time when the "ideal suture" material has been found, the surgeon should depend upon his operative technique to minimize the deleterious affects of the suture material on the healing of a wound. Whether one uses an absorbable or non-absorbable suture is immaterial, providing every known factor regarding the vagaries of the material employed is known to the surgeon. To use a large size catgut when a smaller size will do is asking for complications. The same can be said of any material.

The surgeon should give considerable thought to the making and the closing of an incision. While making the incision the layers of the abdominal wall should be incised cleanly and with sharp instruments. Gentleness in the handling of tissues includes the selection of correct instruments. Whenever possible use eyeless atraumatic needles. For hemostatic purposes, small, straight-bladed, non-toothed clamps are ideal. The hemostatic "bite" should include the bleeding point and, if unavoidable, only a minimum of surrounding tissue. Forceps of the Kocher type with mouse-tooth ends cause excessive trauma and should be seldom used. Pre-operative preparation of the patient with vitamin C will often make it unnecessary to apply forceps to small capillary bleeders. Gauze pressure applied for a few minutes is usually sufficient to produce hemostasis. Sutures in the subcutaneous fat should be avoided, especially catgut; except very fine catgut to suture the subcutaneous fascia.

The fascial layer of an abdominal incision should be incised parallel with the

direction of its fibers. If it becomes necessary to grasp its edges use a non-crushing forceps, such as an Allis—never use a hemostat. Crushed tissue becomes devitalized and interferes with healing. In the closing of the abdominal wound the function of the suture is to hold the tissues together artificially during the "lag period." This period lasts from four to six days; therefore, use a suture material which will accomplish this purpose with a minimum of tissue reaction. Bear in mind that a suture is a foreign body—hence, use the least amount and the smallest size consistent with its holding power over the tensile strength of the tissue to which it is applied.

Placing of two stitches gives only a minor increase in the holding power, whereas with the mattress suture the holding power is increased by 10 to 20 per cent. A mattress stitch, however, requires more material. Shambaugh showed that the strength of a stitch is much greater than the tensile strength of a single strand. He found that the holding power of the tissue and the strength of the stitches are affected directly by the number of stitches placed in a given area. Thus the frequency of stitches depends upon the number required for accurate approximation and upon the required strength of the suture line, for as Shambaugh has shown, the strength of the suture line can be increased only by increasing the number of stitches and not by employing heavier suture material.

A continuous row of suture is stronger than a corresponding suture line with interrupted stitches because the tension is evenly distributed and only two knots are present. On the other hand, the entire strength of the wound is jeopardized when a break occurs anywhere on the continuous suture line. In the event of suppuration, a continuous row gives more trouble than a wound sutured with interrupted stitches. In a continuous suture line more material is utilized which produces greater tissue response.

A figure-of-8 stitch has approximately the same strength as two single stitches; and a mattress stitch is weaker than the figure-of-8 and is no stronger than a simple single stitch. The mattress and Halsted types of stitches are of value for obtaining a wider area of contact between tissues, but as far as the strength of the suture line is concerned a row of simple stitches gives just as satisfactory strength.

One of the most important factors in safeguarding a suture line is the tying of knots. Taylor has made a careful study of knots and concluded that "square knots" and "surgeon's knots" are unreliable for tension sutures when using plain catgut; chromic gut being somewhat more reliable. He advocates that at least a triple throw knot be used in fastening two suture ends. Where three strands enter the knot, as at the end of a continuous suture, a quadruple throw must be used. These conclusions do not apply to small bleeders or where tension on the suture line is not anticipated. Knots made with small size catgut are tied more securely than those with larger sizes. The length of the cut ends isan important factor in the maintenance of a secure knot. The cœfficient of friction, upon which the holding power of the knot is depended, differs according to the material used and whether the material is in a dry state or wet. Serum-proofed and moderately waxed silk, silkworm gut, and dermal do not show any significant variation in the dry or wet state. However, catgut in the moist state almost doubles its coefficient friction, yet we know from experience that wet catgut slips easier than the dry. Taylor explains this by the "elongation" and swelling when catgut becomes moist. Catgut suture ends should be left at least [illegible] mm. long, and cotton and silk ends may be safely left at 3 mm. long.

Accurate approximation of the tissues and avoidance of "dead spaces" are essential for prompt healing. Approximation should be done without puckering, tension, or undue laxity. Wide separation of tissues creates "dead spaces" which soon become filled with exudates that may delay healing, or predispose to infection. When tissues are sutured under tension, the inevitable result is tearing and strangulation. These complications are serious; especially when hollow organs are involved. Finally, bear in mind to avoid suturing tissues of unlike histological structure.

QUESTIONNAIRE

1. Define: (a) ligature; (b) suture.
2. Who introduced antiseptic catgut?
3. Is chemical sterilization of catgut safe?
4. What is the only safe method of sterilizing catgut?
5. What are the requirements for a "perfect ligature"?
6. Give a classification of ligatures.
7. Discuss the uses of the various metallic materials as sutures.
8. Discuss the advantages and disadvantages of the following:
   (a) Silver; (b) vitallium; (c) tantalum; (d) stainless steel; and (e) Michel's metallic clips.
9. What three metals possess sufficient passivity or freedom from electroactivity in the body?
10. What non-absorbable suture materials are derived from animal origin?
11. What is silkworm gut made from?
12. What is its special use?
13. What are the objections to the use of horsehair?
14. How is dermal thread made?
15. How is silk supplied?
16. What are the advantages of silk?
17. Why is silk avoided as an inner row in gastrointestinal anastomoses?
18. Why is it preferred in the presence of carcinoma and for intestinal surgery?
19. What is linen thread made from?
20. How is Pagenstetcher's linen made?
21. What is meant by mercerized cotton?
22. Give the size of catgut for suturing or ligating blood vessels, fasciæ, peritoneum, and for intestinal anastomosis.
23. Would you use cotton as a continuous suture?
24. What is plastigut made from?
25. Discuss the use of nylon as a suture material.
26. How is Kangaroo tendon made? Why is it seldom used?
27. Give the various types of fascial sutures.
28. Where are fascial sutures especially useful?
29. What is McArthur's technique?
30. What is Gallie's technique?
31. Discuss the fate of heterogeneous fascial grafts.
32. In which type of hernia is a heterogeneous graft used?
33. Of what material is catgut made?
34. What is the difference between boilable and non-boilable catgut?
35. What does the size of catgut signify?
36. Give the tensile strength of the various sizes of catgut.
37. What chemicals have been used to render catgut less absorbable?
38. How is the degree of chromitization of catgut designated?
39. What factors alter the absorbability of catgut?
40. Give the loss of tensile strength of catgut when imbedded in tissues.
41. Discuss the use of catgut sutures in fat, peritoneum, muscle, fascia, and for ligating bleeding vessels.
42. Discuss the general principles of "suture technique."

43. When two stitches, or a mattress stitch, are used, is there any increase in the holding power?
44. Discuss the advantages and disadvantages of interrupted stitches over a continuous row of suture.
45. Discuss the coefficient of friction of catgut, silk, and cotton knots.

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CHAPTER 10

KNOTS AND METHODS OF TYING

BY A. V. PARTIPILO

The square knot is for the Surgeon's use; the slipknot is the Hangman's noose.

GENERAL PRINCIPLES

ONE of the most important procedures in surgery is the art of tying a knot securely and with deftness. Tying of knots consumes a major portion of the operating time; hence it behooves a surgeon to be proficient in the art of tying. In surgery, as in all arts and trades, the human element must be taken into consideration. To a tailor, tying and sewing are his means of earning a livelihood, and he cannot hold his position very long if his ties are insecure or if he sews carelessly. A master tailor sews and ties knots neatly and securely, seemingly without effort. His needles pass in and out at regular intervals, without loss in motion and with a momentum which has a fascination of its own. The same is true of all master artisans; their movements are rhythmical, seemingly without effort, and executed flawlessly.

The surgeon rarely sews with the same simplicity of a tailor, or ties knots with security and ease of a sailor. Many tie with no forethought, and as a result the knot is either a "granny" or more often a slip knot. The consequence of this haphazard method of tying is an insecure knot which may slacken or slip off entirely. Undoubtedly this has been many times the cause of postoperative hemorrhage and death. In these cases life may truly be said to hang on the fate of a single thread.

To tie a knot properly, with just sufficient strength, requires much practice. The surgeon should practice ceaselessly until he has developed a faultless technique, tying a knot easily, quickly, and securely. It is also a decided advantage to learn, and be proficient in a variety of methods in order to cope with the different conditions while operating. In this manner more time will be saved and the knots will be tied with greater security.

Simple Knot.—A simple knot, or "half-hitch," is one in which the material is looped and one end is twisted completely around the other, thus making a single throw or half-hitch (Fig. 20). A simple knot is the first maneuver in making of a square or granny knot. It is seldom employed alone for tying purposes, except in ligating small capillary bleeders in the superficial tissues, when security is not essential.

Square Knot.—A square (reef, flat, or true) knot is a double simple knot, the second "half-hitch" made in the reverse direction of the first so that the two stretches of the cord pass together under the loop formed by the second half-hitch (Fig. 20). The square knot is the accepted method of tying, and when properly made it can be relied upon.

Granny Knot.—A granny knot is made by superimposing one simple knot upon another without reversing the second half-hitch. The two stretches of the cord do

however, the hands must be crossed and traction made in the plane of the loop (Fig. 24, *b*).

3. *Traction in the Plane of the Loop.*—As illustrated in Figures 23, *b* and 24, *b*, the two half-hitches of a square knot are completed by making divergent traction in the plane of the loop. Failure to do this will result in the formation of a slip knot.

Thus, when tying a square knot there are three important maneuvers which must be strictly adhered to: namely, (1) the two half-hitches are made in a reverse manner; (2) the cords are crossed in one of the turns, whereas the hands are crossed in the other; and (3) to prevent the formation of a slip knot, traction must be made in the plane of the loop.

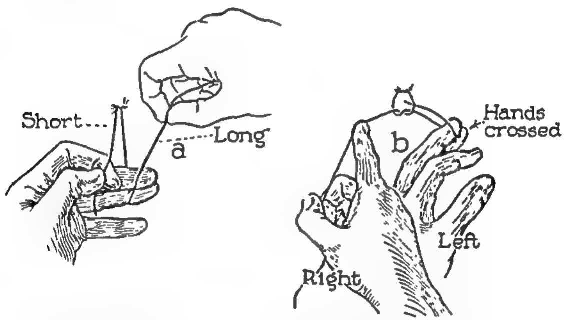


FIG. 24.—Position of the hand and cord in the second half-hitch: (*a*) the knot is begun without crossing of the ligature; (*b*) in order to pull in the plane of the loop, the hands are crossed upon completion of the knot.

Methods of Tying Knots.—There are numerous methods of tying a square knot, and it is a decided advantage for a surgeon to become proficient with various methods. Different localities and situations call for different methods. The author has found that for the great majority of cases the one-hand method is practical, quick, and effective. The surgeon should practice until all the movements are coördinated reflexly and the tying is performed effortlessly and without conscious thought. With constant practice and persistent effort other methods can be mastered equally well.

One-hand Method.—Figures 25 and 26 (pages 129 and 130) illustrate the entire series of movements for tying a square knot with one hand. When the free end of the ligature lies on the right side, the knot is begun with the cord lying on the palmar surface as illustrated in Figure 25, *a*. When the short end lies on the left, the knot is begun with the cord held on the dorsal surface as shown in Figure 26, *a*.

Following are descriptions and illustrations for each individual maneuver:

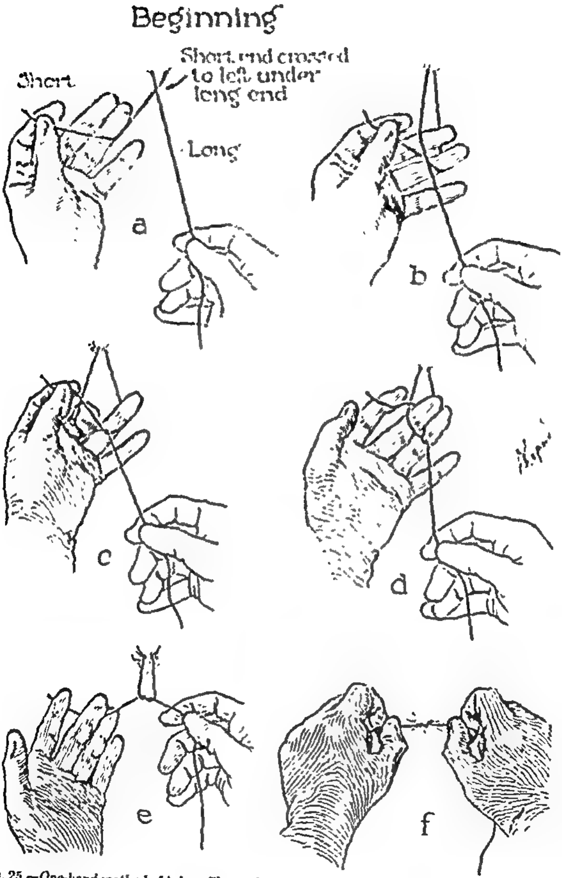


FIG. 25.—One-hand method of tying Shows the various maneuvers for making the first half-hitch.

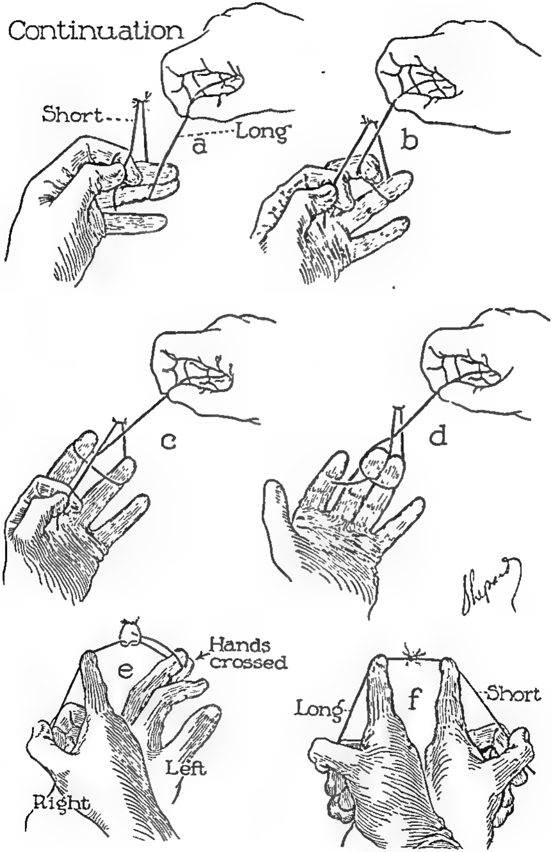


FIG. 26.—One-hand method of tying. Second half-hitch.

The First Half-hitch (Figs. 25 and 27–32)

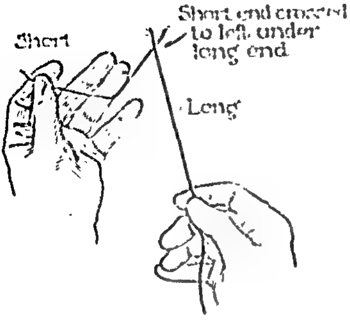


FIG. 27

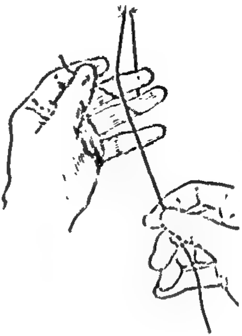


FIG. 28

FIG. 27.—The short end is grasped between the thumb and index finger, with the cord lying on the palmar surface of the middle and the third finger. The long end is held with the right hand. Notice that in this initial step the cord is crossed with the short end beneath the long end.

FIG. 28.—The next step consists of bringing the long end over the dorsum of the middle finger of the left hand.

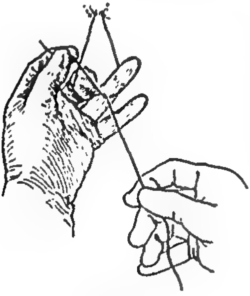


FIG. 29

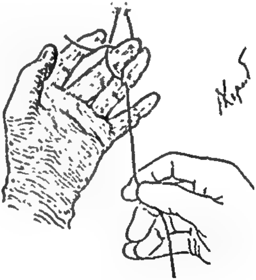


FIG. 30

FIG. 29.—The middle finger is now flexed until its dorsal surface catches the short end held by the thumb and index finger. During this maneuver the third finger is held in extension and is not flexed at any time.

FIG. 30.—Extend the middle finger, thus grasping the short end between the middle and third fingers. At the same time the short end held by the thumb and index finger is released.

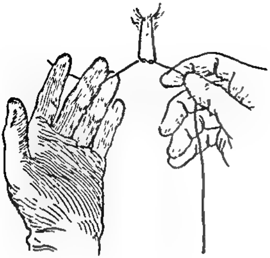


FIG. 31

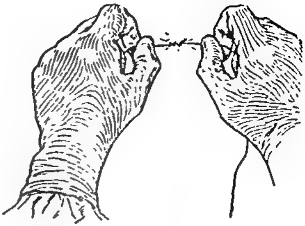


FIG. 32

FIG. 31.—The short end is now pulled through the loop by the middle and third finger.

FIG. 32.—The final step of the first half-hitch consists of tightening the knot by making divergent traction in the plane of the loop.

The Second Half-hitch (See Figures 26 and 33-38)

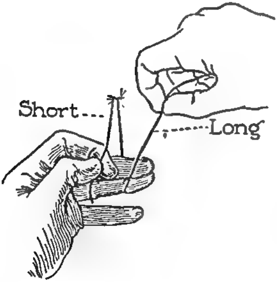


FIG. 33

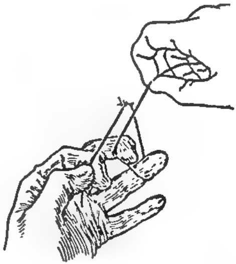


FIG. 31

FIG. .33—The short end is held over the dorsum of the middle finger and grasped by the thumb and index finger. The long end of the ligature is brought beneath the third finger and over the palmar surface of the left hand.

FIG. 34.—With the ligature held taut, the middle and third fingers are flexed until they lie behind the short end held by the thumb and index finger.

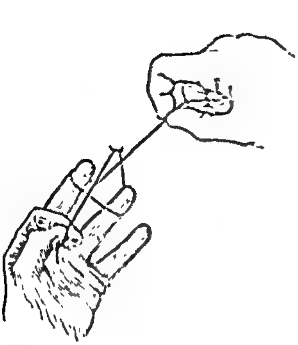


FIG. 35

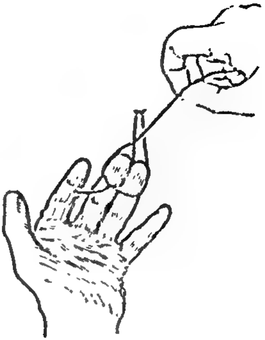


FIG. 36

FIG. 35.—With an upward motion the middle finger is brought above the short end; then, by extending the two fingers, the cord is made to lie between them.

FIG. 36.—The short end is grasped tightly between the middle and index fingers and pulled through the loop.

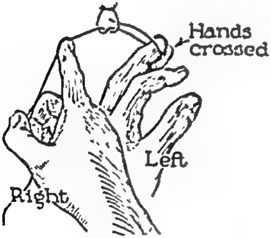


FIG. 37

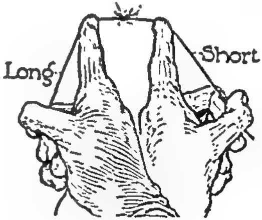


FIG. 38

FIG. 37.—In order to apply traction in the plane of the knot, the hands are crossed.

FIG. 38.—The second half-hitch is now completed by tightening the knot, applying divergent traction with the hands crossed. Notice the position of the fingers.

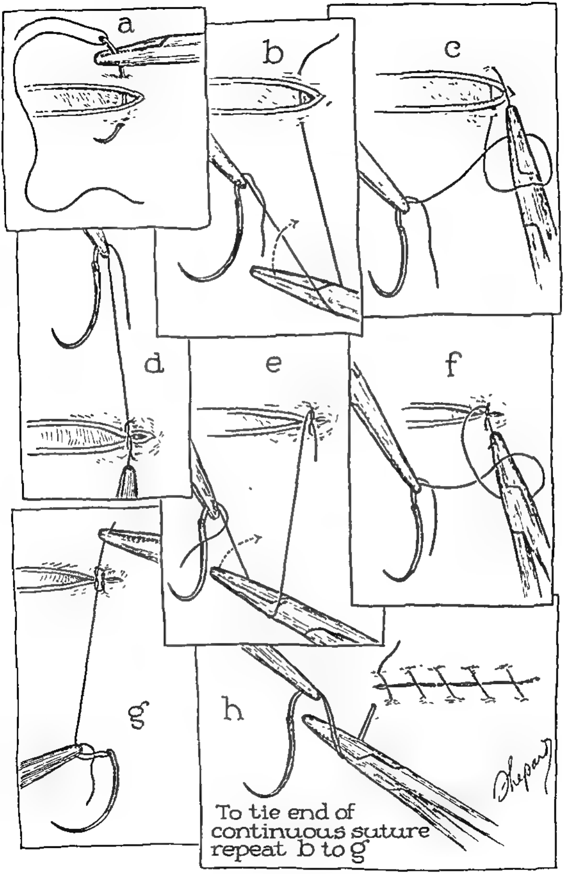


FIG. 39.—Grant's "No-Hand-Touch-Technique": *a* to *d*, first-half hitch; *e* to *g*, second half-hitch

Grant's "No-Hand-Touch-Technique" for Tying Knots.—There are many occasions when Grant's method for tying knots offers a rapid, economical, and practical method of tying. It has especial advantages when tying in deep cavities, and for "no-hand-touch-technique" in orthopedic operations.

Procedure: 1. After the tissues have been transfixed (Fig. 39, *a*), the needle is pulled through until the short end is only about one inch from the point of fixation. The long end is toward, whereas the short end is away from the operator.

2. The point of the needle holder is placed over the long end (Fig. 39, *b*), and the latter is then looped around the needle-holder.

3. With the suture thus looped, the needle-holder grasps the short end (Fig. 39, *c*). At the same time the long end is pulled in a direction away from the operator (Fig. 39, *d*), thus making divergent traction in the plane of the knot.

4. The second half-hitch is begun with the needle-holder placed beneath the long end of the suture. The remaining maneuvers are similar to those described above, except that they are in the reverse direction. (See Figure 39, *e* to *g*.) Note that in the second half-hitch divergent traction is made in the plane of the knot with the short and long ends pulled in the opposite direction.

Two-hand Method of Tying.—The two-hand method of tying, as illustrated in Figures 42 and 43 (pages 136 and 137), is practical and especially effective when the long end of the suture is found to the left of the operator. The tying can be made around the fingers of either hand; however, the left hand is generally employed. This method of tying is useful when firm and continuous pressure is required during the whole process of tying. It can also be readily used when the ligature material is short.

The First Half-hitch (See Figures 42 and 40–49)

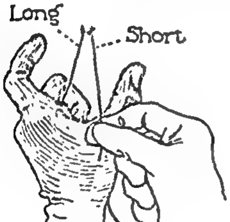


Fig. 40

Fig. 41

Fig. 40.—The long end of the ligature is grasped with the second, *third*, and little finger of the left hand; the index finger and thumb are widely separated and extended. The short end held by the *thumb and index finger* of the right hand, is brought between the extended thumb and index finger of the left hand.

Fig. 41.—The thumb of the left hand is now placed over the short end and beneath the long end.

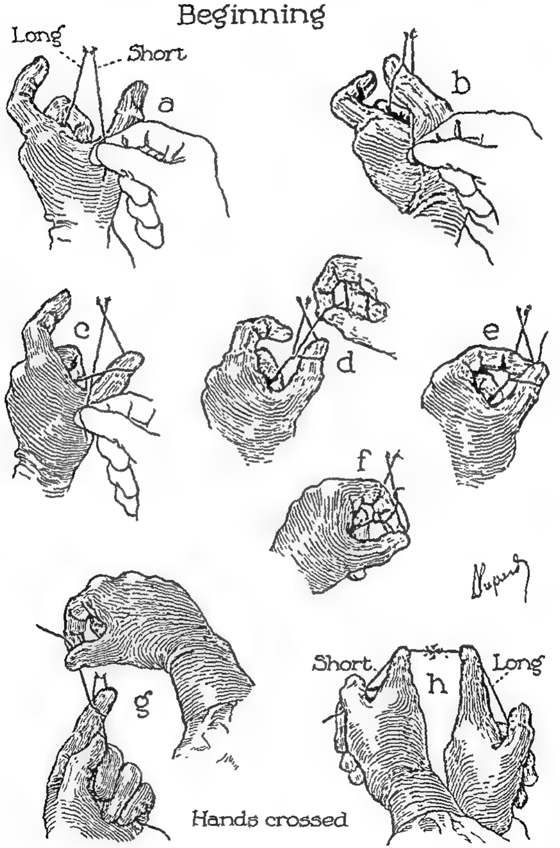


Fig. 42.—Two-hand method of tying. First half-hitch.

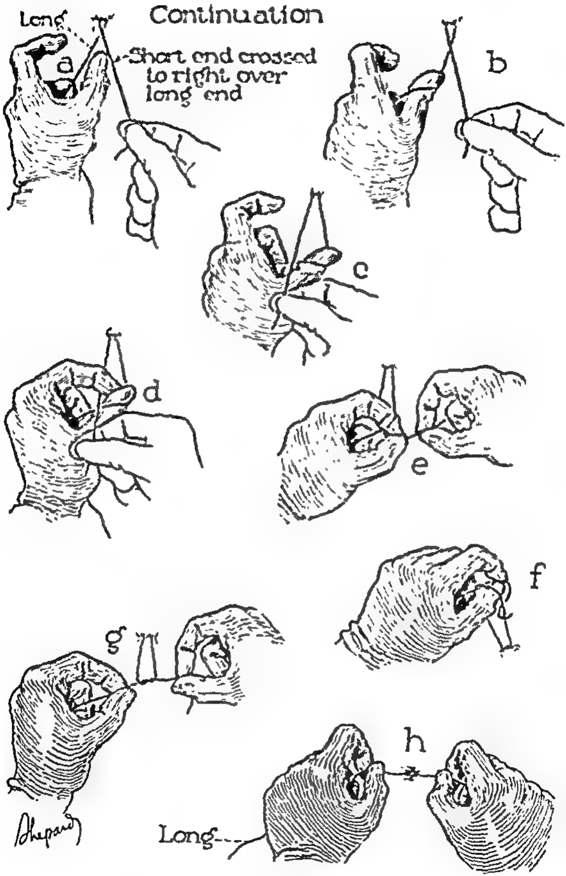


FIG. 43.—Two-hand method of tying. Second half-hitch.

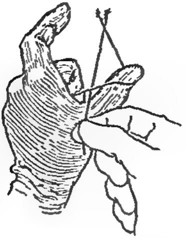


Fig. 41

Fig. 44.—Now extend the thumb, thus crossing the long end over the short end; at the same time a loop is produced.

Fig. 15

Fig. 45.—At this time bring the short end on the palmar surface of the thumb.

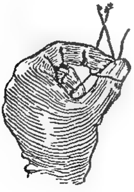


Fig 46

Fig. 47

Fig. 46.—Grasp the short end between the tips of the index finger and thumb of the left hand, at the same time releasing it from the right hand.

Fig. 47.—With the short end thus held, the loop is made to fall on the index finger, and the short end is then carried through the loop.

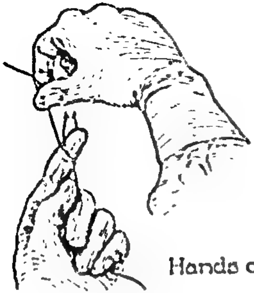


FIG. 48

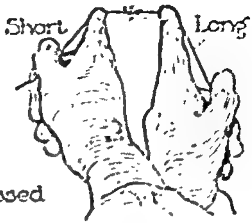


FIG. 49

FIG. 48.—After the short end has been pulled through the loop, it is grasped by the right hand.

FIG. 49.—Traction is made by crossing the hands, thus making divergent pull in the plane of the loop. When the first half-hitch was begun, the long end was to the left of the operator; when the knot is completed, however, it is found at his right.

The Second Half-hitch (Figures 43 and 50-57)

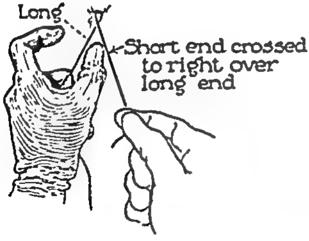


FIG. 50

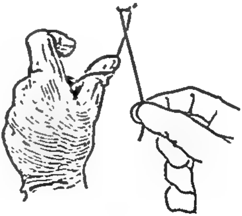


FIG. 51

FIG. 50.—The long end is grasped with the left hand, leaving the thumb and index finger outstretched and free. The short end is brought over the long end. Notice that the cord is crossed with the short end over the long end.

FIG. 51.—With the ligature held as described above, the thumb of the left hand is entwined over the long end by successively flexing and extending the thumb.

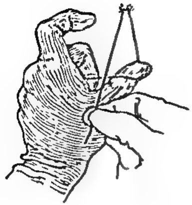


Fig 52

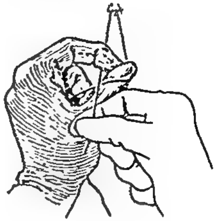


Fig. 53

Fig. 52.—The short end is now brought between the index finger and thumb, thus producing a loop.

Fig. 53.—The tip of the index finger is approximated to the tip of the thumb.

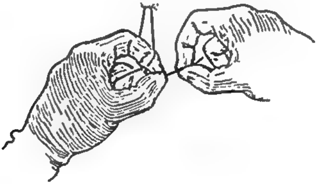


Fig. 54

Fig. 54.—By supinating the left hand, the loop is made to fall on the index finger. The short end of the ligature is now grasped by the left index finger and thumb.

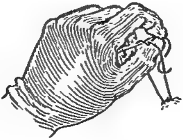


Fig 55

Fig. 55.—The short end is released by the right hand and pulled through the loop by the index finger and thumb of the left hand. This is done by pronating the hand, thus introducing the thumb through the loop.

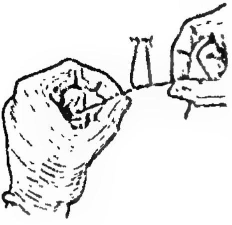


FIG. 56

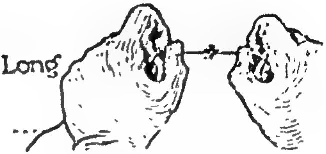


FIG. 57

FIG. 56.—The short end is grasped and traction is made in the plane of the loop

FIG. 57.—Method of tightening the knot.

Murphy's "No-Hand-Touch-Technique."—Murphy's no-hand-touch-technique and Grant's method for tying knots are especially adaptable for orthopedic surgery. The Grant method has the advantage in that a knot can be made with a shorter ligature than by Murphy's method, it is also more useful when tying in deep cavities.

Figure 58 illustrates the various steps in making a square knot. While the assistant holds the short end perpendicular, the operator grasps the long (needle) end with a tissue forceps and with a forceps held in the right hand a loop is produced as illustrated in Figure 58, *a*. The long (needle) end is now grasped with the right hand forceps (Fig. 58, *b*) and pulled through the loop. The knot is then completed by making divergent traction in the plane of the loop, as illustrated in Figure 58, c. The second half-hitch is made in a reverse direction, as illustrated in Figures 58, *d*, *e*, and *f*.

QUESTIONNAIRE

1. Discuss the importance of developing a good tying technique.
2. Define: Simple knot, square knot, granny knot, slip knot, and surgeon's knot.
3. What is a reef knot?
4. What are the objections to a granny knot?
5. Why is a slip knot dangerous to use?
6. How is a square knot converted into a slip knot?
7. How is a surgeon's knot made?
8. What are the advantages and disadvantages of a surgeon's knot?
9. What three important steps are employed in tying a square knot?
10. Why should traction be made in the plane of the "loop"?
11. Why should a surgeon learn more than one method of tying?
12. What are the advantages of "One-hand" methods of tying?
13. When is Grant's "No-Hand-Touch-Technique" used?
14. When is Murphy's method of tying used?

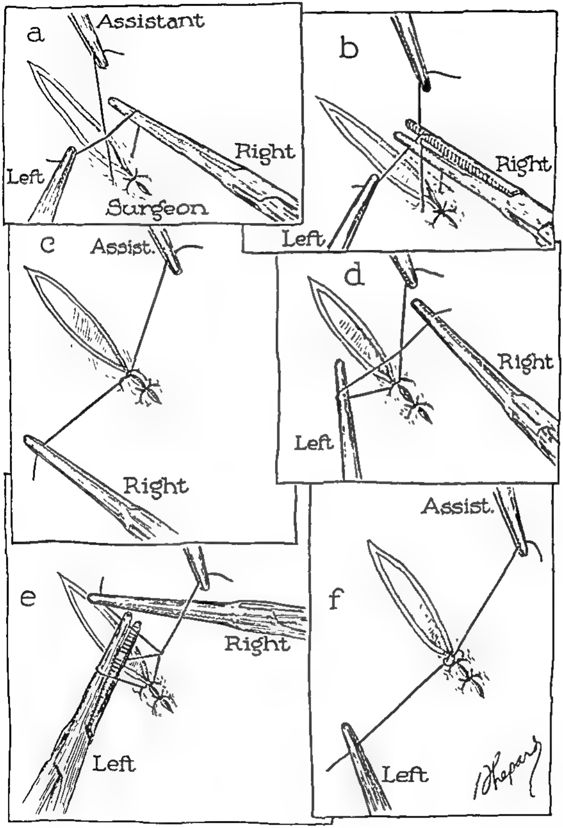


FIG 58 —Murphy's "No-Hand-Touch-Technique"; *a* to *c*, first half-hitch; *d* to *f*, second half-hitch.

CHAPTER II

SURGERY OF THE NECK—ANATOMY

By A. V. Partipilo and S. W. Osborn

SURFACE ANATOMY AND LANDMARKS

THE skin of the neck differs from that of other parts of the body in that it is under voluntary control through its attachment and close relationship with the underlying platysma muscle. This muscle, the platysma myoides, is a thin, broad, fan shaped sheet extending from the pectoral region of the chest to the face. It arises from the deep fascia of the pectoral region and from the clavicle, extends upward and medialward to become inserted into the inferior border of the mandible and then becomes continuous with the triangular and inferior quadratus muscles. The anterior fibers decussate with the opposite platysma below and behind the chin. From the center of this, the fibers of the muscle slope downward to the sterno-clavicular junction. Thus the midline of the neck and the lowest part of the anterior triangle is uncovered.

The superficial fascia of the neck carrying the superficial vessels and nerves, is rather thin and is closely attached to the platysma, however, it is cleanly separated from the deep cervical fascia by a distinct cleavage thus permitting free mobility of the skin. The chief interest of the platysma to the surgeon is that in closing an incision of the skin, the muscle must be sutured if he wishes to avoid a large scar which may result from traction by the unsutured muscle.

Landmarks of the Neck.—On the anterior and lateral surfaces of the neck are a number of prominences, muscles, and vessels which can either be seen or palpated by the examiner's hand. These are of surgical importance and every surgeon should be familiar with them. As illustrated in Figure 59, the following landmarks are located on the anterior surface of the neck:

1. Laryngeal Prominence of the Thyroid Cartilage.—With the head held in extreme extension, the most prominent landmark is the laryngeal prominence popularly called "Adam's apple." In relation to the cervical vertebra it is situated at about the level between the 4th and 5th vertebræ. The right and left laminæ of the cartilage can be felt on either side of the prominence and by careful manipulation the superior cornu can be palpated. The short and thick inferior horn cannot be so easily detected. The level of the vocal folds corresponds to the middle of the anterior border of the thyroid cartilage. When performing a thyrotomy the surgeon should be careful to incise the thyroid cartilage exactly in the midline in order to avoid injuring the vocal folds.

2. The Superior Thyroid Notch.—This is found immediately above the laryngeal prominence and is formed by the V-shaped separation of the laminæ.

3. The Hyoid Bone.—By flexing the head slightly the body of the hyoid bone can be palpated one or two centimeters above the laryngeal prominence. It is situated at the level of the 3rd cervical vertebra. During the act of swallowing the

occipital and subclavian triangles. In the median line of the neck is the submental triangle located just above the hyoid bone.

Digastric Triangle.—The digastric or submaxillary triangle is bounded above by the lower border of the body of the mandible and an extension of this plane as represented by the stylomandibular ligament. Below, the triangle is bounded by the posterior belly of the digastric and in front, by the anterior belly of the digastric muscle. The stylomandibular ligament, a continuation of the deep cervical fascia, extends from the styloid process to the posterior border of the mandible to divide the digastric triangle into an anterior and a posterior portion. In the anterior portion is the submaxillary gland which is overlapped by the posterior half of the mandible and extends to the great horn of the hyoid bone (Fig. 63). The gland is

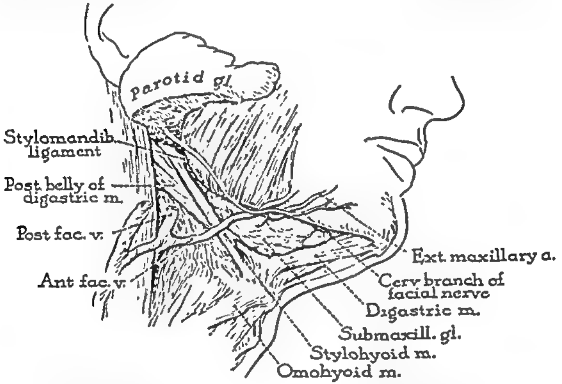


FIG 63.—Digastric triangle. Note the position of the submaxillary gland and its relationship with the anterior facial vein and the cervical branch of the facial nerve.

separated from the parotid by the stylomandibular ligament. The posterior compartment is occupied by the parotid gland. The floor of the digastric triangle is formed, from the front backward, by three flat muscles: the mylohyoid, hyoglossus, and the superior constrictor of the pharynx (Fig. 64). The submaxillary gland conceals a great part of these muscles.

As illustrated in Figure 63, the anterior facial vein is found anterior to the gland, while the external maxillary artery is seen deep in relation to the gland and arches upward over the mandible. The cervical branch of the facial nerve, after leaving the under surface of the parotid gland, follows the mandibular border of the submaxillary gland and crosses in front of the facial vein and external maxillary artery. The external maxillary artery commences in the carotid triangle separate or in common with the lingual artery (Fig. 64). It is situated deep to the posterior belly of the digastric and the stylohyoid muscles and the submaxillary gland. The hypoglossal nerve enters the carotid triangle beneath the digastric and stylohyoid

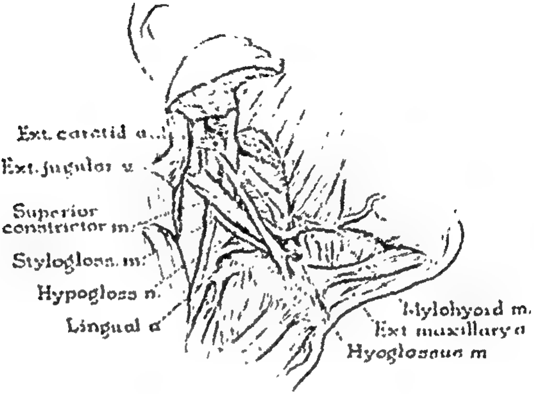
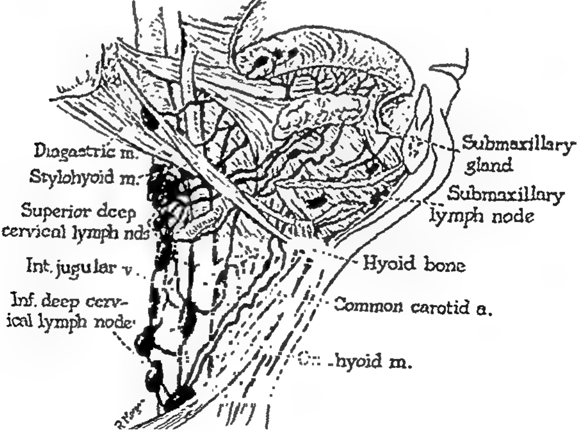
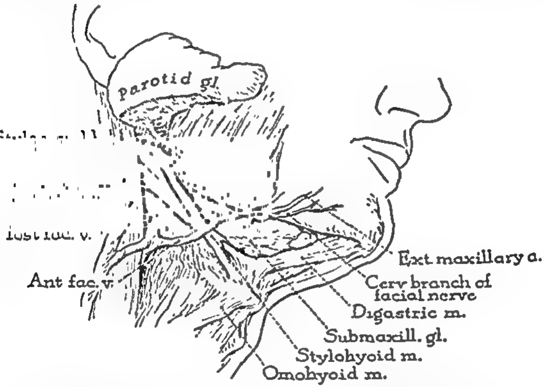


Fig. 64.—Deeper dissection of the digastric triangle. The sternocleidomastoid muscle has been divided and the submaxillary gland has been removed.



occipital and subclavian triangles. In the median line of the neck is the submental triangle located just above the hyoid bone.

Digastric Triangle.—The digastric or submaxillary triangle is bounded above by the lower border of the body of the mandible and an extension of this plane as represented by the stylomandibular ligament. Below, the triangle is bounded by the posterior belly of the digastric and in front, by the anterior belly of the digastric muscle. The stylomandibular ligament, a continuation of the deep cervical fascia, extends from the styloid process to the posterior border of the mandible to divide the digastric triangle into an anterior and a posterior portion. In the anterior portion is the submaxillary gland which is overlapped by the posterior half of the mandible and extends to the great horn of the hyoid bone (Fig. 63). The gland is



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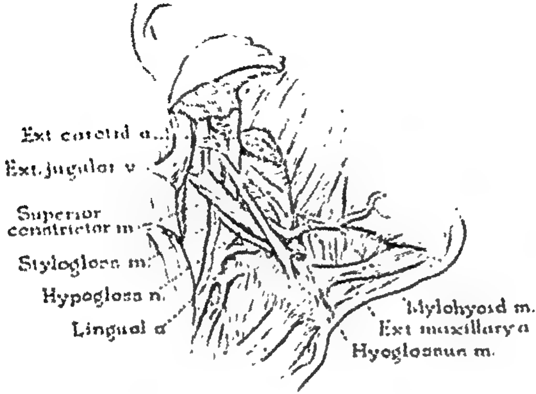

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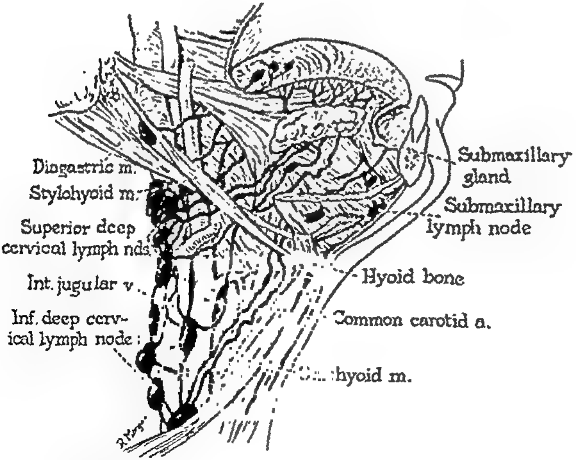

Fig. 65.—Deep lymphatic lymph nodes in the digastric and carotid triangles. Lymph drainage of the tongue is shown. Note the mode of communication between the various nodes. (After Eycleshymer and Jones.)

muscles, then passes anterior to the external maxillary and lingual arteries and continues forward as far as the tip of the tongue.

The submaxillary lymph nodes, three to six in number, lie in the angle beneath the lower border of the body of the mandible and on the anterior surface of the submaxillary gland (Fig. 65). The most constant of these, the middle node of Stahr, lies on the external maxillary artery as it courses over the mandible. Rarely, some smaller nodes are found on the deep surface of the submaxillary gland.

The afferents of the submaxillary lymph nodes carry lymph from the medial palpebral commissure, from the lateral aspect of the nose, the upper lip, the lateral part of the lower lip, the anterior third of the tongue, the gums, the submaxillary and sublingual glands and adjacent portions of the floor of the mouth.

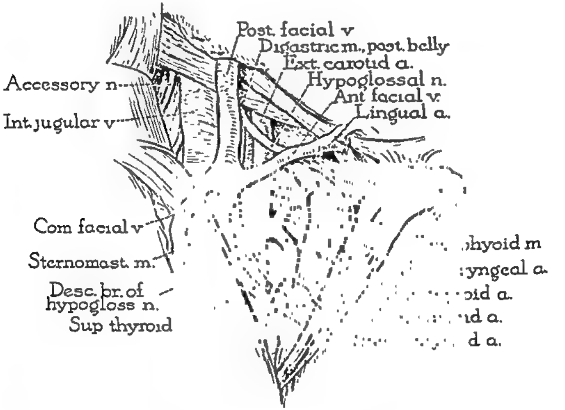

FIG. 66 —Carotid triangle. Superficial dissection to show the relationship of the vessels and nerves within the triangle.

Carotid Triangle.—The carotid triangle is bounded from behind, by the anterior border of the sternocleidomastoid muscle; below, by the the superior portion of the omohyoid muscle, and above, by the posterior belly of the digastric and stylohyoid muscles, and the great horn of the hyoid bone forming the apex. The floor of the triangle is formed by the thyrohyoid, hyoglossus, and the medial and inferior constrictor muscles.

The posterior belly of the digastric muscle forming the superior boundary of the carotid triangle, holds a key position in the anatomy of this region and for this reason it should be studied in all its details. It arises, under cover of the sternocleidomastoid muscle, from the mastoid notch on the medial side of the mastoid process. It is directed downward and forward, accompanied by the stylohyoid, to end in an intermediate tendon attached to the great horn of the hyoid bone. The mastoid process and the overlying sternocleidomastoid muscle conceals the origin

of the digastric muscle and the submaxillary gland courses over it. Aside from these coverings, it is superficially placed and only three structures are found anterior to it. These are the tributaries of the common facial vein, the cervical branch of the facial nerve, and the great auricular nerve. Note that the remaining great nerves and vessels of the neck pass deep to the muscle.

The contents of the carotid triangle may be conveniently grouped under three headings: (1) Nerves: *accessory, hypoglossal, and vagus.* (2) Vessels: internal jugular vein, common carotid artery, and the internal and external carotid arteries. (3) Lymph glands.

As illustrated in Figure 66, in order to expose the various structures within the carotid triangle, the deep cervical fascia is incised along the anterior border of the sternocleidomastoid muscle and the latter is retracted outward. The *accessory nerve*, the spinal portion of the XI nerve, is seen emerging from under the digastric muscle and after crossing the internal jugular vein it passes obliquely downward and laterally toward the sternocleidomastoid where the nerve disappears under cover of the muscle at about 1½ to 2½ inches below the tip of the mastoid. In this region the accessory nerve is surrounded by the superior deep cervical lymph nodes and is accompanied by the sternomastoid branch of the occipital artery. The nerve continues downward and laterally and makes its appearance in the posterior triangle of the neck at about the junction of the upper and middle third of the posterior border of the sternocleidomastoid muscle.

The second nerve in the carotid triangle is the hypoglossal. This nerve enters the triangle underneath the posterior belly of the digastric then turns medially, loops around the occipital artery and passes forward anterior to the external carotid and lingual arteries then to disappear underneath the digastric tendon and continue forward to the tip of the tongue where it supplies its intrinsic muscles.

The descending hypoglossal is a long slender branch of the hypoglossal nerve. It arises from the hypoglossal just as the latter crosses the occipital artery. It passes downward underneath the sternocleidomastoid muscle and on the superficial surface of the carotid sheath. Just above the omohyoid muscle it is joined by the 2nd and 3rd cervical nerves to form the ansa hypoglossal. Figure 67 is a schematic illustration to show the make up of the ansa hypoglossal nerve, hypoglossal, vagus, etc., and their connections.

The vagus is the third important nerve in the carotid triangle. It descends within the carotid sheath posterior to and between the carotid vessels and the internal jugular vein as far as the sternoclavicular joint where it takes a different course on either side of the neck. On the right side it runs anterior to the subclavian artery and behind the innominate vein where it gives off the *recurrent* laryngeal nerve (*see* Chapter 12 on Thyroid). The left vagus descends between the left common carotid and subclavian arteries deep to the left innominate vein. It crosses the left side of the aortic arch and passes downward to reach the dorsal aspect of the root of the lung. The left recurrent laryngeal nerve springs from the vagus as the latter crosses the aortic arch.

Aside from the recurrent laryngeal, the vagus gives off the following branches *in the neck:* the pharyngeal, superior laryngeal, and superior cardiac branches. The pharyngeal, usually two in number, receives its motor fibers *through* the ganglion nodosum from the accessory nerve. It passes obliquely downward between the internal and external carotids and joins the pharyngeal branch of the glossopharyngeal to form the pharyngeal plexus on the wall of the pharynx at the

level of the middle constrictor muscle. It supplies all the voluntary muscles of the pharynx and soft palate except the tensor palati and stylopharyngeal muscles. The nerves of the carotid body are filaments from the glossopha[illegible] ... [illegible] ...ssibly

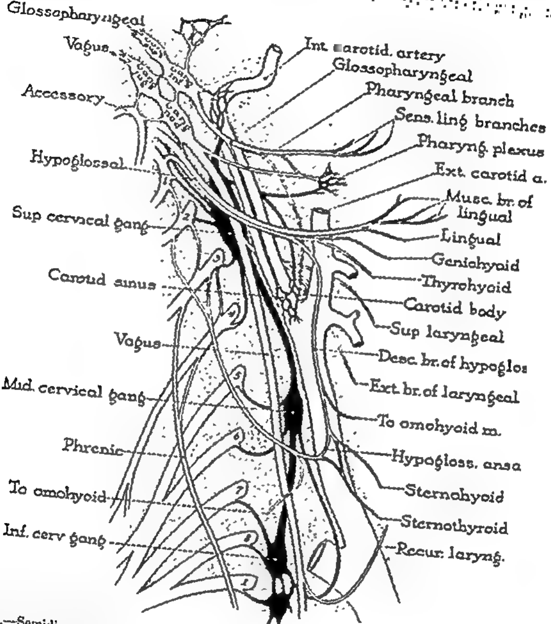

FIG. 67.—Semidiagrammatic drawing illust... thetic nerves and the formation ... carotid artery. (After Gray's A...

The ... nodos... and n... cartilage where it divides ...carotid artery toward the middle constrictor muscle branch proceeds deep to the sternohyoid muscle to supply motor fibers to the cricothyroid muscle and part of the inferior constrictor muscle. The external branch passes anteriorly to the thyrohyoid membrane to supply sensory fibers to the larynx and vocal cords. It sends filaments along the superior thyroid artery and ends at the inferior horn of the thyroid cartilage.

The superior cardiac branches, two or three in number, arise at variable parts of the vagus. Very often they are derived from the superior laryngeal nerve. They pass along the common carotid artery to the aorta where they help to form the cardiac plexus.

Another nerve which may be of surgical importance is the carotid nerve which is located on the internal carotid artery and ends at the carotid sinus at the bifurcation of the common carotid. It arises from the main trunk of the glossopharyngeal and communicates with the ganglion nodosum by way of the pharyngeal nerve. Sometimes this nerve is denervated when medical treatment fails to relieve a patient with a carotid sinus syndrome.

The second important structure in the carotid triangle is the internal jugular vein. It is a direct continuation of the transverse sinus and begins at the jugular foramen at the base of the skull. It descends vertically downward, at first behind the internal carotid artery but, as it reaches the root of the neck, it becomes more superficial and gradually assumes an anterior position and overlaps the common carotid especially on the left side. In this area the right vein passes in front of the first part of the subclavian artery and its branches and on the left side it is anterior to the terminal part of the thoracic duct. The vein enters the carotid triangle by passing deep to the stylohyoid and digastric muscles. At this point the vein is crossed superficially by the accessory nerve and the sternomastoid branch of the occipital artery. The hypoglossal nerve lies just medial to it.

In the neck, the tributaries of the internal jugular vein are the common facial, the lingual, pharyngeal, superior thyroid, and middle thyroid veins (Fig. 68). The common facial has two tributaries, the anterior and posterior facial. The anterior facial takes a course similar to the external maxillary artery, except that the artery is deep to the posterior belly of the digastric while the vein is superficial to the muscle. The posterior facial vein arises in front of the ear through the union of the superficial and medial temporal veins. It runs doward underneath or in the substance of the parotid gland and deep to the facial nerve. It finally enters the neck and joins the anterior facial to form the common facial which is situated at a point corresponding to the level of the hyoid bone. The common facial vein and its tributaries is of importance in surgery of the parotid and submaxillary glands and in the dissection of the superior lymph nodes in the digastric and carotid triangles.

A collateral circulation is established by a branch of the posterior facial vein communicating with the external jugular, and a twig from the anterior or common facial with the anterior jugular vein.

The lingual vein which returns blood from the tongue follows a tortuous course along the lingual artery. It enters the carotid triangle and empties into either the common facial, superior thyroid or the internal jugular vein.

The pharyngeal veins vary in number and come from the pharyngeal plexus from the pharynx and larynx and end in the upper part of the internal jugular vein. However, they may occasionally open into the facial, lingual, or the superior thyroid vein.

The superior thyroid vein has its origin in the substance of the thyroid gland. From its point of origin the vein travels along the superior thyroid artery to end in the internal jugular vein below that of the common facial. The superior thyroid vein has communicating branches with the inferior thyroid and common facial veins.

The third important structure in the neck is the common carotid artery and

its branches. The artery, largest in the neck, has a different point of origin and is of unequal length on the contralateral sides. The right carotid has its origin at the bifurcation of the innominate artery posterior to the right sternoclavicular articulation. The left carotid, arising from the highest part of the arch of the aorta, has a thoracic and a cervical portion, thus being longer than its mate on the right. The relationship of the two vessels in the neck is practically the same, except that the thoracic duct does not come into relationship with the right carotid. The left recurrent laryngeal nerve crosses posterior to the thoracic part of the left carotid

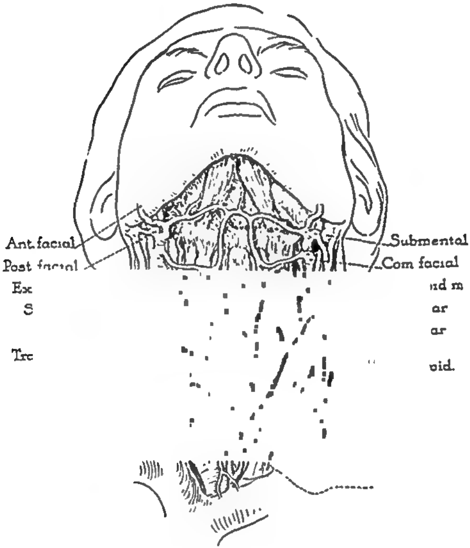

FIG. 68.—Deep and superficial veins of the neck. Illustrates the venous collateral circulation.

and medial to its cervical part, while the right recurrent passes posterior to the lower part of the right artery to reach its medial side. The internal jugular vein on both sides lies lateral to the artery. In the lower part of the neck, the left vein is anterior to the artery, while on the right it is separated at the root of the neck, from the lateral surface of the artery in an interval where the vagus nerve is seen.

The common carotid artery, enclosed within the carotid sheath, ascends upwards, laterally and backwards and, at the level of the highest part of the thyroid cartilage, it divides into the external and internal carotid arteries (Fig. 69). The two branches ascend side by side, the internal being postero-lateral, the external,

anteromedial, and pass deep to the posterior belly of the digastric muscle to enter the digastric triangle. The internal carotid artery gives off no collateral branches in the neck so that the blood supply to the various viscera of the neck is supplied by the external carotid and partly by the inferior thyroid artery. From its point of origin, the external carotid ascends upward and laterally toward the neck of the mandible where it divides into two terminal branches, the superficial temporal and internal maxillary arteries. In the carotid triangle the external carotid gives off the following branches: The superior thyroid, lingual, external maxillary, occipital, and ascending pharyngeal.

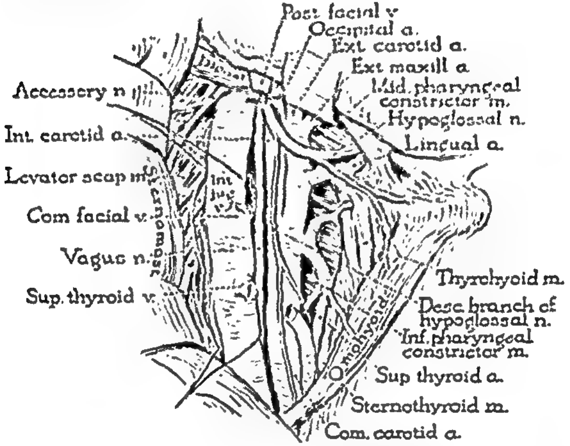

FIG. 60.—Deeper dissection of the carotid triangle illustrating the various branches of the carotid artery and structures within the carotid sheath.

The superior thyroid artery is the first branch of the external carotid and arises just after the bifurcation of the common carotid. This artery is described in greater detail in Chapter 12.

The lingual artery arises at a point between the origins of the superior and external maxillary arteries, at the level of the great horn of the hyoid bone. The first part of the artery is superficial and covered by skin, platysma and deep cervical *fascia*. It rests upon the constrictor pharyngis muscle. At first it runs upward and medialward then it curves downward, crossed by the hypoglossal nerve, and passes deep to the posterior belly of the digastric to enter the digastric triangle. This artery is the only vessel supplying the tongue.

The external maxillary artery, also known as the *facial*, arises from the external carotid just above the lingual artery. From its point of origin the artery takes a tortuous course; it passes upward underneath the posterior belly of the digastric and posterior to the submaxillary gland, then it turns around the lower border of

the mandible, pierces the deep fascia and becomes rather superficial. It ends at the angle of the mouth where it becomes the angular artery. The artery is superficial in its beginning and at its terminal portions. In the carotid triangle it is covered by skin, platysma, and deep cervical fascia whereas, after it turns over the lower border of the mandible, it is covered only by skin and platysma. In the interval it is covered by digastric muscle and the submaxillary gland. The branches of the external maxillary may be divided into two groups: (1) cervical branches; *ascending palatine, tonsilar, glandular, submental and muscular;* (2) *facial* branches: inferior labial, superficial labial, lateral nasal, angular and muscular.

The occipital artery arises from the posterior surface of the external carotid at a point opposite the external maxillary artery. From its point of origin, it is directed upward and posteriorly, running parallel and underneath the posterior belly of the digastric muscle to terminate near the medial end of the superior nuchal line of the occipital bone and in the superficial fascia of the posterior part of the scalp. Close to its point of origin, the artery is crossed by the hypoglossal nerve, however as it ascends it successively crosses the internal carotid artery, the vagus nerve, the internal jugular vein and accessory nerve. It is covered by the sternocleidomastoid muscle and later it is under cover of the posterior belly of the digastric muscle.

The ascending pharyngeal arises from the medial side of and near the origin of the external carotid artery. It ascends on the inferior, middle and superior constrictor muscles as far as the base of the skull. It supplies the pharynx, soft palate and meninges.

The lymph nodes in the carotid triangle are part of the chain of nodes in the neck called the deep cervical lymph nodes. They are found in close relationship with the internal jugular vein from the base of the skull to the clavicle. They are conveniently divided by the omohyoid muscle into two general groups: (1) the superior deep cervical lymph nodes, and (2) the inferior deep cervical lymph nodes. Each group is further subdivided into a medial and a lateral group.

The medial group of the superior nodes lies on the superficial surface of the internal jugular vein in the carotid triangle. The highest of these lies in the area of the union of the common facial vein with the internal jugular vein and are concerned with drainage from the tongue, lips, gums, cheeks and outer part of the nose. Another group is closely associated with the accessory nerve so that this nerve is frequently sacrificed in radical neck dissections. The lowest nodes in this group are located on the internal jugular vein just above the omohyoid muscle. This group is also known as the jugulo-omohyoid nodes.

The superior group of lymph nodes receive efferents from all parts of the head and neck through the following sub chains: (1) Infra-hyoid group consisting of: *a*, those located on the thyro-hyoid membrane whose efferents follow the superior laryngeal artery and come from the vocal cords and larynx; *b*, a pre-laryngeal group which are inconsistent except those found on the cricothyroid ligament; *c*, a pre-thyroid group occasionally found near the superior and inferior margins of the thyroid isthmus, and *d*, a para-tracheal group found between the esophagus and trachea along the inferior thyroid artery and the recurrent laryngeal nerve. (2) The suprahyoid group of nodes are those found above the hyoid bone in the area bounded by the mandible above, the hyoid bone below, and the anterior belly of the digastric muscle laterally. The submental nodes are located superficial to the mylohyoid muscles and between the anterior bellies of the two digastric muscles.

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They receive afferents from the gums, tip of the tongue, and the skin beneath the chin and their efferents pass partly to the submaxillary lymph nodes and partly to the deep cervical glands. The submaxillary lymph nodes, three to six in number, lie under the deep fascia of the neck in the angle between the lower border of the mandible and the submaxillary gland. The afferents are from the side of the nose, the upper lip, the lateral part of the lower lip, the anterior third of the border

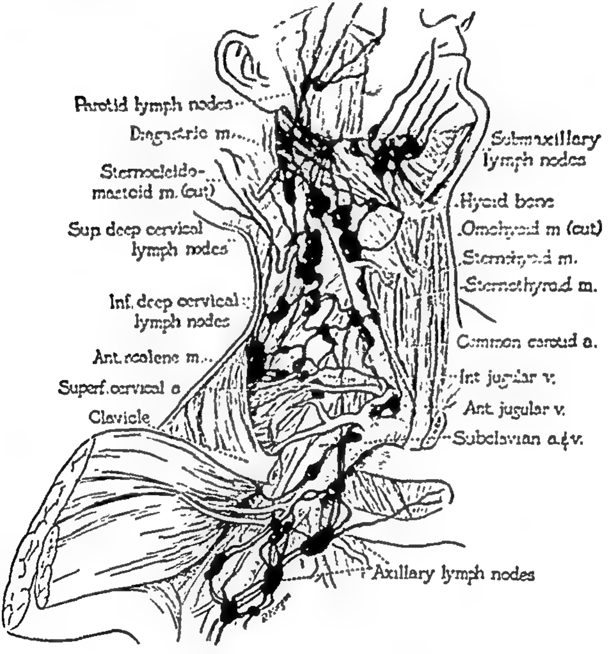

Fig. 70.—Lymphatic drainage of the neck.

of the tongue, the gums, the submaxillary and sublingual glands and the adjacent parts of the floor of the mouth. The efferents terminate in the upper deep cervical nodes.

The inferior deep cervical lymph nodes, also called the supraclavicular nodes are found in the muscular triangle below the omohyoid muscle. They extend beyond the margin of the sternocleidomastoid muscle into the supraclavicular

triangle where they are in relation with the brachial plexus and the subclavian vein. This group of glands, the lateral nodes, receive efferents from the lower part of the neck, from the upper part of the thorax, and from the deep parts of the mammary glands and axilla. Their efferents join the jugular lymphatic trunk. The medial group of nodes are in relationship with the lower part of the internal jugular vein. They receive efferents from the upper medial group and send efferents to the jugular lymph trunk.

The Muscular Triangle.—This triangle is also known as the vertebral-arterial triangle, the prevertebral-intermuscular triangle and the inferior carotid triangle. It is bounded in front by the midline of the neck, behind by the anterior belly of the omohyoid muscle above, and the anterior margin of the sternocleidomastoid muscle below. The floor is made up by the longus coli and anterior scalenus muscles. The apex of the triangle is the prominent tubercle of the sixth cervical vertebra.

Beginning anteriorly, the following structures are encountered in posterior progression: the skin, the superficial fascia, platysma, the deep cervical fascia, and under cover of these are the sternohyoid and sternothyroid muscles, the thyroid gland and its isthmus, the larynx, and trachea, the carotid sheath containing the internal jugular vein, the common carotid artery and the vagus nerve, the esophagus, the vertebral vessels, and the thoracic duct. The surgical anatomy of this triangle is further described in Chapter 12 on surgery of the thyroid gland.

Posterior Triangle.—The posterior triangle of the neck is bounded by the sternocleidomastoid muscle in front, by the anterior margin of the trapezius muscle behind; its base is formed by the middle third of the clavicle, and its apex is at the occipital bone. The posterior belly of the omohyoid muscle as it crosses between the sternocleidomastoid and trapezius muscle at about 1 inch above the clavicle, divides the triangle into the occipital and subclavian triangles.

The floor of the triangle is formed by a group of muscles whose fibers run obliquely downward and backward. From above downward, these muscles are, the splenius capitis, levator scapulæ, and the three scaleni muscles. The scalenus medius arises from the posterior tubercles of the transverse process of the lower six cervical vertebræ. The muscle descends along the side of the vertebral column and becomes inserted into the upper surface of the first rib between the groove for the subclavian artery and the neck of the rib. The scalenus posterior arises from the lower two or three posterior tubercles of the transverse processes of the cervical vertebra and becomes inserted to the outer surface of the second rib. The scalenus anterior lies deep in the neck behind the sternocleidomastoid muscle. It arises from the anterior tubercles of the transverse processes of the third to the sixth cervical vertebræ and is inserted into the scalene tubercle of Lisfranc, which is situated on the inner border of the first rib between the groove for the subclavian vein in front and the groove for the subclavian artery behind. The lateral border of the first rib forms the lower limit of the floor where the subclavian artery becomes the axillary artery.

The roof of the posterior triangle is formed by the deep cervical fascia which covers the sternocleidomastoid and the trapezius muscles. This is the same cervical fascia encountered on the anterior surface of the neck. The floor of the triangle is covered by the prevertebral fascia which forms a covering for the subclavian vessels and the cervical part of the brachial plexus.

The subclavian vein and artery, and the brachial plexus are the important

structures in the triangle. The subclavian vein begins at the lateral border of the first rib and ends at the medial border of the anterior scalenus muscle by joining with the internal jugular vein to form the innominate vein. The scalenus muscle separates the subclavian vein from the subclavian artery. It also divides the subclavian artery into three parts. The subclavian vein has one tributary, the external jugular vein. This vein descends on the deep cervical fascia from the angle of the jaw obliquely downward and backward and pierces the deep fascia at the posterior border of the sternocleidomastoid muscle an inch or so above the clavicle and ends nto the subclavian vein.

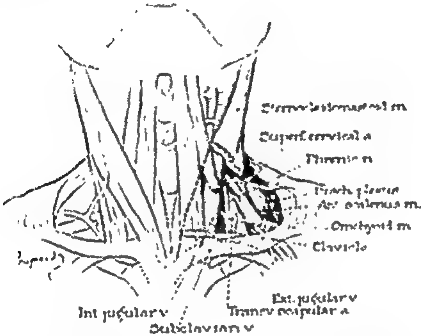

FIG. 71.—Regional anatomy for phrenic nerve interruption.

The phrenic nerve lies on the ventral surface of the scalenus anterior, crossing the muscle from its lateral to its medial border. It is under cover of the sternocleidomastoid muscle and, in the subclavian triangle, it continues with the scalenus muscle as it enters the thorax. To expose the nerve, an incision is made about one inch above the clavicle, the middle part of which is the lateral border of the sternocleidomastoid muscle. This muscle is mobilized and retracted medially and in developing the dissection, some fibers of the posterior border of the muscle may be cut. The external jugular vein is mobilized and retracted laterally. After the sternocleidomastoid muscle has been retracted a pad of fat is seen covering the scalenus muscle; this is trangressed and the scalenus is visualized. The phrenic nerve is then seen coursing downward and medially on the muscle.

As illustrated in Figure 73, the brachial plexus has its origin in the neck from the anterior primary divisions of the fifth, sixth, seventh, eighth cervical nerves, and the first thoracic nerve. The roots from these nerves emerge between the anterior and middle scaleni muscles, above the third part of the subclavian artery. From these roots three trunks are formed namely; (1) The upper or superior trunk formed by the union of the fifth and sixth cervical nerves; (2) the middle trunk formed by the seventh cervical alone; and (3) the inferior trunk formed by the

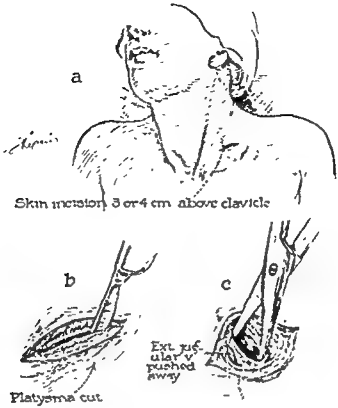

FIG. 72.—Incision for exposure to phrenic neurectomy.

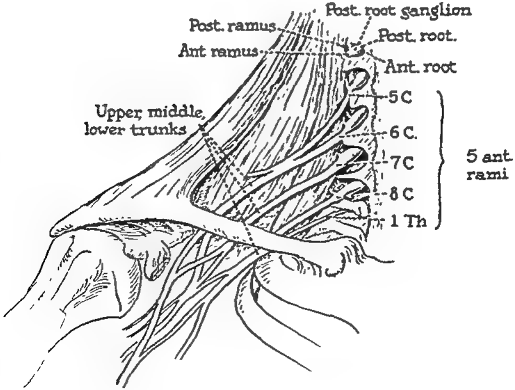

FIG. 73.—Cervical trunks of the brachial plexus

eighth cervical and first thoracic nerves. The trunk enter the posterior triangle from behind the lateral border of the anterior scalenus muscle and lie upon the medius muscle. The inferior trunk is found behind the subclavian artery and lies on the first rib, thus being actually outside of the posterior triangle.

After a short distance, the trunks split into an anterior and a posterior division and from these arise three cords which are located in the axilla. The three cords are termed the lateral, medial and posterior, named according to their relation to the axillary artery.

The relationship of the inferior trunk to the first rib and of the subclavian artery to the anterior scalenus muscle and the first rib is of surgical importance. Scalenus anticus syndrome is a clinical entity which may result from compression of the plexus and the subclavian artery due to abnormal elevation of the first thoracic rib or its fibrous prolongation, as a result of spasm and shortening of the anterior scalenus muscle.

QUESTIONNAIRE

1. How does the skin of the neck differ from that of other parts of the body?
2. Describe the extent of the platysma muscle.
3. What is the chief interest of this muscle to the surgeon?
4. Give the various landmarks found on the anterior surface of the neck.
5. What is "Adam's Apple"?
6. What is the anatomical level of the vocal cords? Give its surgical significance.
7. Through which membrane is the epiglottis reached in subhyoid laryngotomy?
8. Where is the usual anatomical site in suicidal "cut throat"?
9. What is the relationship of the thyroglossal duct with the hyoid bone?
10. Discuss the relationship of the isthmus of the thyroid to the tracheal rings.
11. What is meant by high and low tracheotomy?
12. Why is a low tracheotomy avoided?
13. What is another name for jugular notch?
14. Give the various important structures found in the vicinity of the jugular notch.
15. Give the various landmarks found on the lateral surface of the neck.
16. What is the carotid tubercle? Describe method in locating it.
17. What is another name for carotid tubercle and what is its importance?
18. Describe the vertebral triangle.
19. Give the various structures found within this triangle.
20. What is the surgical significance of the midpoint of the sternocleidomastoid muscle?
21. Draw a diagram illustrating the various triangles of the neck.
22. Bound the digastric triangle.
23. What important structures are found in the anterior digastric triangle?
24. What important structures are found in the posterior triangle?
25. Bound the carotid triangle.
26. Give the various nerves and vessels found in this triangle.
27. Describe the relationship and course of the accessory nerve.
28. Describe the relationship and course of the hypoglossal nerve.
29. How is the ansa hypoglossal nerve formed?
30. Give the relationship and course of the vagus nerve.
31. Give the various branches of the vagus nerve.
32. What is the origin of the carotid nerve?
33. Why is it of surgical importance?
34. Give the course and relationship of the internal jugular vein.
35. What are its tributaries?
36. Give the cour
37. Give the vari
38. Where is the

39. Classify the deep cervical lymph glands.
40. Bound the muscular triangle.
41. What structures are found within this triangle?
42. Bound the posterior triangle.
43. What important structures are found within this triangle?
44. Give the relationship and course of the phrenic nerve.
45. Describe the method of exposing this nerve.
46. What part of the brachial plexus is found with this triangle?
47. What is the Scalenus anticus syndrome?

Chapter 12

SURGERY OF THE NECK

THYROIDECTOMY

By A. V. Partipilo

GENERAL CONSIDERATIONS

The thyroid is a ductless gland developed from a median proliferation of the ventral wall of the pharynx. The median outgrowth extends downwards and its lower end bifurcates to form the isthmus and the lateral lobes of the thyroid; the upper end gives rise to the foramen cecum of the tongue. The intervening portion is the thyroglossal duct which is canalized during fetal life but normally disappears later in life. When the duct fails to become completely obliterated, accessory thyroids or thyroglossal cysts may develop. These may occur anywhere along the duct and even at the back of the tongue. A thyroglossal cyst is usually connected by means of the duct with the foramen cecum, hence its removal requires the removal of this connection otherwise recurrence is apt to take place.

The gland substance is composed of cuboidal epithelial cells arranged in a single layer around spheroidal, oval, or polygonal spaces known as alveoli, acini or vesicles. The alveoli contain a homogeneous gelatinous material, the colloid substance, which is the stored secretion of the gland. The gland is surrounded by a translucent connective tissue membrane, the true capsule, which sends prolongations inward to form a framework for the gland tissue and dividing the gland into lobules and finally giving support to the alveoli. The alveolar connective tissue conveys blood vessels, lymphatics and nerves to the gland.

The thyroid gland possess an internal secretion which is functionally closely related for the normal growth of young individuals and for maintaining normal metabolism throughout life. It has been known for a long time that there is a definite relationship between the iodine content and the histological structure of the thyroid gland, and that iodine is an essential element of the thyroid hormone. Marine, Williams and Lenhart, in 1908, found that hyperplasia and hypertrophy of the gland are due to a deficiency of iodine and that the amount stored in the gland varies directly with the amount of colloid and inversely with the degree of active hyperplasia. The total store of iodine in the gland is from 10 to 15 mg. According to Marine, when the content is below 1 mg. per gram of dried gland (normal content, 2 mg. per gram of dried gland) the gland is in an abnormal state; in hyperthyroidism it may be as low as 0.25 mg. or less per gram of tissue. He was of the opinion that all thyroid enlargements, including the toxic types, result from the demands made upon the gland to produce its hormone without adequate supply of iodine. The exact nature of the thyroid hormone is not definitely known. Baumann, in 1896, was the first to demonstrate that the thyroid gland normally contains iodine. In 1899, Oswald showed that the active iodine constituent was attached to a protein, globulin, found in the colloid of the acini. Kendall in 1914

isolated the active principle of the thyroid gland and called it thyroxin. It contains about 65 per cent of iodine. This substance has the same physiological properties of dessicated thyroid gland. It functions as a catalytic agent in the process of oxidation of the tissues. One milligram of thyroxin elevates the basal metabolism 2.8 per cent.

The prophylactic use of iodine in the prevention of goiter was started in the public schools of Akron, Ohio, in 1917, by Marine and Kimball. Loe and Zondek in 1921, showed that administration of potassium iodide reduces the metabolic rate in exophthalmic goiter and, in 1923, Plummer introduced Lugol's solution as a preoperative treatment in toxic goiters. In 1943, Astwood reported the use of thiouracil in the treatment of hyperthyroidism. Many investigators have since carried out clinical investigations with varying results. The principle action of thiouracil is to prevent the synthesis of thyroxine by the thyroid. Because of toxic reaction this drug is not widely used. At present, propyl thiouracil is being used and early reports indicate that it is less toxic than thiouracil. It is approximately 5 times as effective as thiouracil. The initial dose of propyl thiouracil is 75 mg. per day and followed by a maintainance dose of from 25 to 50 mg. daily. Curtis and Swenson[1] in an excellent and extensive survey of the literature and from their studies of patients in their clinic, made the following conclusions pertaining to the use of thiouracil-like drugs:

1. The drugs are capable of causing severe toxic reactions, and should not be used unless accurate observations can be made frequently, preferably under hospital conditions.

2. The incidence of leucocytic depression is higher than is generally recognized. The depression of circulating leucocytes has thus far been interpreted to be due to depression of the bone marrow elements responsible for their production, rather than to any effect upon the mature leucocytes per se.

3. The development of agranulocytosis is detectable in the bone marrow hours before it is shown by a drop in the circulating leucocytes, and similarly, the decrease in the circulating leucocytes will occur before clinical symptoms develop.

4. Toxic reactions will also occur during the treatment with propyl thiouracil.

5. The presence of nodular goiter should make one hesitant regarding the employment of thiouracil-like drugs.

6. The drugs appear to be contraindicated by the following:

a. Allergic conditions·

(1) Previous history of multiple allergies to food, drugs, and to foreign protein.

(2) Previous history of sensitivity to the sulfonamide drugs. Simultaneous sulfonamide therapy appears inadvisable.

(3) Previous history of toxic reactions to one of the thiouracil-like drugs.

b. Bone marrow conditions:

(1) Pre-existing low initial white count.

(2) Pre-existing blood dyscrasias due to bone marrow hypoplasia.

(3) The presence of an infection demands that the leucocyte response be optimal, and few will risk of unpredictable development of agranulocytosis in such cases

[1] Curtis, George M., and Swenson, Roy E. Thiouracil and its allies in the treatment of hyperthyroidism, an experimental and clinical survey. Surg. Gynec. & Obst., 86, 105–123, Feb., 1948

c. Exophthalmos, if severe, appears to contraindicate the use of the drugs.
d. Adolescence; long continued treatment is inadvisable at this time.
e. Toxic nodular goiter; long continued is inadvisable if this condition exists. The threat of malignancy development is well recognized. The functioning fetal adenoma falls into the same category.
f. Pre-existing liver disease.
g. Pre-existing adrenal disease.
h. Pre-existing renal disease.
i. Pregnancy.
j. Large cervical, retrosternal, retrosternal and retrovascal, and retrovisceral goiters, as pressure symptoms may result in these conditions.

7. Indications for the use of the thiouracil-like drugs appear to be found within that group of patients in whom the risk of thiouracil therapy is less than the risk of the disease, when other methods of control are used, *i.e.*, patients presenting:
a. Refractoriness to iodine.
b. Hypersensitivity to iodine.
c. Recurrent hyperthyroidism after previous thyroidectomy.
d. Recurrent hyperthyroidism with hypertension.
e. Ordinarily bad risks for preoperative preparation:
(1) Thyrocardiacs.
(2) Thyrocachetics.
f. Hyperthyroidism, and patients who cannot for one reason or another have surgery.

SURGICAL ANATOMY

The thyroid gland is situated anterio-laterally to the thyroid and cricoid cartilages, vertically over the fifth and sixth tracheal rings and extends backward to the larynx. It is composed of two lobes connected by the isthmus which lies across the second, third and fourth tracheal rings. The gland lies underneath the superficial muscles of the neck and is surrounded by a layer of the deep cervical fascia which forms the surgical capsule of the thyroid gland. The inferior surface of the thyroid gland is convex and its lower point is at about the fifth or sixth tracheal rings near the superior border of the sternum. Enlarged glands may be located beneath the sternum or the clavicle (retrosternal, retrocervical) or may lie within the thoracic cage (intrathoracic goiters). The superior pole of the thyroid gland extends upward and backward to the posterior border of the thyroid cartilage. The posterior borders of the lobes are in contact with the esophagus and lower part of the pharynx, while posteriorly they partially overlie the carotid sheath.

Muscles Concerned in Thyroidectomy.—The muscles concerned in surgery of the thyroid gland are those found within the anterior triangle on the lateral aspect of the neck. The lateral aspect of the neck is divided into a posterior and an anterior triangle by the sterno-mastoid muscle. It arises by two heads; (1) the sternal head, and (2) a broad clavicular head from the superior surface and medial third of the clavicle. The muscle is attached by a short tendon to the lateral surface of the mastoid process and to the superior nuchal line. The sterno-mastoid muscle is rather prominent and can be felt along its entire extent. The external jugular vein can be seen to cross the muscle at about 1 centimeter below its mid-portion

1 centimeter above the external jugular vein at the lateral aspect of the sternocleidomastoid muscle. Immediately underneath and medial to the sternocleidomastoid muscle are found the infrahyoid (ribbon) muscles: omohyoideus, sternohyoideus, sternothyroideus and thyreohyoideus. These muscles are enclosed within the deep layer of the superficial fascia of the neck. They overlie the thyroid gland. The ribbon muscles may be cut across when the gland is large, when the superior pole is highly located, and when the gland is found underneath the sternum or in the thoracic cavity.

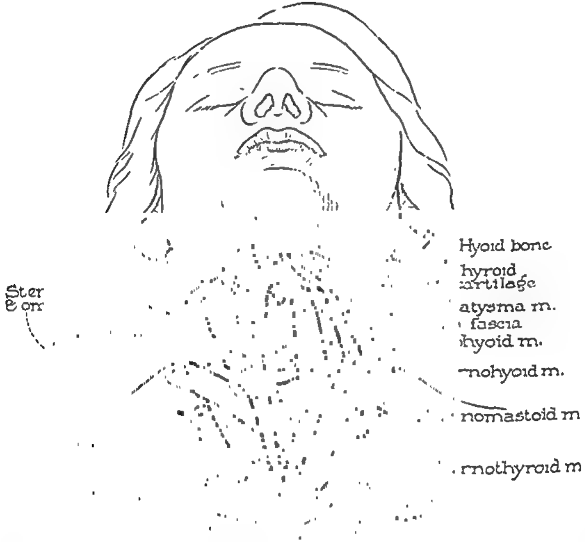

FIG 74 —Muscles concerned in thyroidectomy.

The Surgical Capsule.—In the neck the deep fascia consists of two layers; (1) a superficial, and (2) a deep layer. The superficial layer is further subdivided into two layers; a superficial layer which encloses the sternomastoid muscle and is traced backwards over the posterior triangle of the neck to surround the trapezius. The deeper layer of the superficial fascia invests the ribbon muscles. The deep fascial layer is a continuation of the superficial layer. It forms an enveloping sheath (carotid sheath) for the common carotid artery, the internal jugular vein, and the vagus nerve. From the carotid sheath a layer proceeds antero-medially to the thyroid gland. This is known as the pretracheal fascia or surgical capsule of the thyroid gland Posteriorly it is connected with the prevertebral fascia. The surgi-

cal capsule is separated from the thyroid gland by the true or anatomical capsule. In the space between the two capsules are found the arterial and venous vessels supplying the gland. The anatomical capsule is distinguished from the surgical capsule by its firm and intimate connection with the glandular substance. Thus, it cannot be stripped from the gland without tearing the parenchyma.

The two lobes of the thyroid gland are enclosed in separate fascial layers. The isthmus is usually contained in one of the fascial layers, although it may have a separate compartment. There is a reduplication of this fascia at the superior pole of the thyroid gland where it is perforated by the superior thyroid artery, and this cone-like arrangement should be well dissected free when ligating the superior vessel or pole.

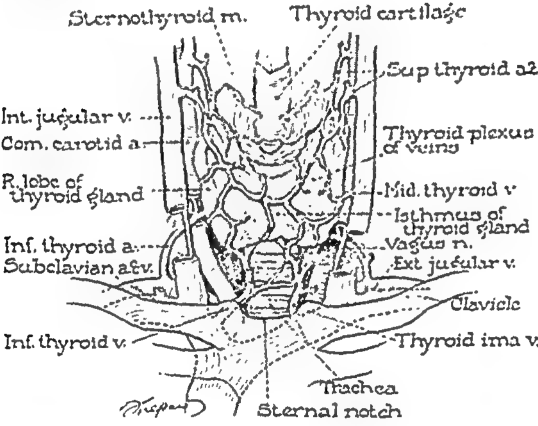

FIG. 75.—Blood supply of the thyroid gland.

Blood Vessels of the Thyroid Gland.—The thyroid gland is the most vascular organ in the body. The arterial vessels enter through the surgical capsule, divide into smaller branches, and finally form a dense capillary network that surrounds the follicles. There are usually four vessels, the respective right and left superior and inferior thyroid arteries. The thyroidima, present in about 8 per cent, is a small artery arising from the arch of the aorta, perforating the surgical capsule and entering the gland on its inferior surface.

The superior thyroid artery is the first branch of the external carotid artery. It arises just above the bifurcation. Occasionally, it has its origin from the common carotid artery, just before the bifurcation. From its origin, the artery ascends parallel with the great cornu of the hyoid bone; passes under the omo-hyoid and sterno-thyroid muscles perforates the surgical capsule to reach the superior pole of the gland where it lies anterior to the gland. The artery immediately divides into

1 centimeter above the external jugular vein at the lateral aspect of the sternocleidomastoid muscle. Immediately underneath and medial to the sternocleidomastoid muscle are found the infrahyoid (ribbon) muscles: omohyoideus, sternohyoideus, sternothyroideus and thyreohyoideus. These muscles are enclosed within the deep layer of the superficial fascia of the neck. They overlie the thyroid gland. The ribbon muscles may be cut across when the gland is large, when the superior pole is highly located, and when the gland is found underneath the sternum or in the thoracic cavity.

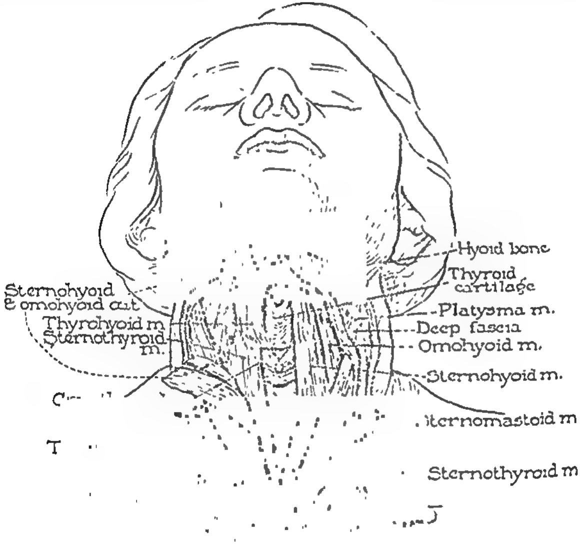

FIG. 71.—Muscles concerned in thyroidectomy.

The Surgical Capsule.—In the neck the deep fascia consists of two layers; (1) a superficial, and (2) a deep layer. The superficial layer is further subdivided into two layers; a superficial layer which encloses the sternomastoid muscle and is traced backwards over the posterior triangle of the neck to surround the trapezius. The deeper layer of the superficial fascia invests the ribbon muscles. The deep fascial layer is a continuation of the superficial layer. It forms an enveloping sheath (carotid sheath) for the common carotid artery, the internal jugular vein, and the vagus nerve. From the carotid sheath a layer proceeds antero-medially to the thyroid gland. This is known as the pretracheal fascia or surgical capsule of the thyroid gland. Posteriorly it is connected with the prevertebral fascia. The surgi-

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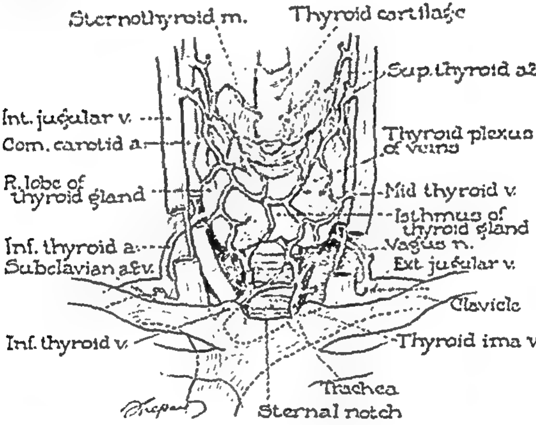

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three branches to supply the thyroid gland; (1) the external branch located along the external border of the lobe; (2) the internal branch which anastomoses with its counterpart on the opposite side; and (3) the posterior branch which passes between the thyroid gland and the trachea. These vessels anastomose freely with the inferior thyroid artery. There is usually a perforating branch which gives some supply to the surgical capsule. The superior thyroid artery should be ligated before it enters the superior horn of the gland since it divides just before entering the substance of the gland.

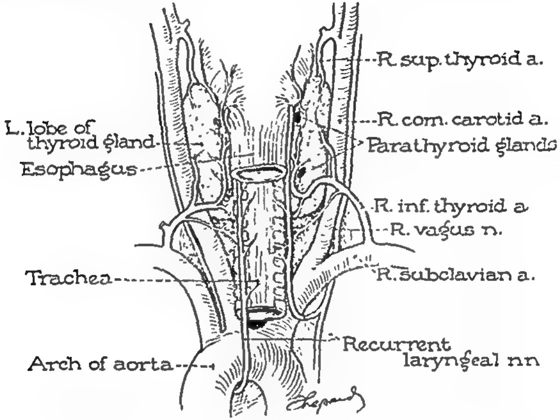

FIG. 76.—The relationship of the recurrent laryngeal nerve and the parathyroid bodies to the posterior-medial surface of the thyroid gland.

The inferior thyroid artery is a branch of the thyroid axis; the latter has its origin from the subclavian. From its point of origin the artery ascends vertically to within one-half inch of the carotid tubercle (the sixth cervical vertebra) and at this point it curves sharply medially and downward to enter the gland on its lateral inferior aspect.

There are three principle veins which convey blood from the thyroid gland, as follows: (1) The superior thyroid group follows the course of the superior thyroid artery and empties into the internal jugular vein. (2) The middle thyroid vein begins at the lateral border of the gland and follows the inferior thyroid artery to empty into the internal jugular vein. (3) The inferior thyroid vein descends directly downward and empties into the innominate vein.

The Recurrent Laryngeal Nerve.—In the region of the neck each recurrent laryngeal nerve ascends in the groove between the trachea and esophagus. The right recurrent nerve arises at the root of the neck; as the vagus nerve crosses in front of the first part of the subclavian artery (*see* Fig. 76) the recurrent hooks around the subclavian and passes upward and medially behind or in front of the

inferior thyroid artery. It continues upward in the groove between the esophagus and the trachea. It finally disappears beneath the inferior border of the inferior constrictor muscle and ends in supplying the muscles of the larynx. The left recurrent nerve arises from the vagus as it crosses the aortic arch. After hooking around the arch it passes upwards in the neck where its relationship and course is similar to that of the right nerve. The right nerve is more often in front of the inferior thyroid artery. For this reason this nerve is more apt to become injured when ligating the inferior pole of the gland. In relation to the thyroid gland the recurrent nerve lies on the posterior-medial aspect of the gland and is placed outside of the surgical capsule. Hence in thyroidectomy the posterior-medial portion of the gland and capsule are left behind to avoid injuring the nerve.

ANESTHESIA

Only local anesthesia will be described because this is the method which we generally use. General anesthetics may be the method of choice in certain conditions and in children. In the non-toxic goiter the condition of the patient permits any type of anesthesia. However, even in these the use of local anesthesia has its distinct advantages which may be briefly stated as follows: (1) Permits the patient to exercise the voice thereby giving the surgeon a means of determining the status of the recurrent laryngeal nerve; (2) The risk of pulmonary complications is materially reduced since it permits the patient to clear mucus from the bronchi during or immediately after the operation; (3) Postoperative general affects are minimized; and (4) if necessary nitrous oxide may be used temporarily at certain stages of the operation without unduly affecting the patient's general condition.

One of the most important pre-requisite to a good local anesthesia is the absolute confidence of the patient that the anesthesia will be done without pain. For this reason the surgeon should be absolutely positive that his technic is faultless. In our operating room, the word Pain is never mentioned. There is nothing which will make a patient nervous and apprehensive than to ask her if the insertion of the needle or a maneuver is causing pain. If you ask a patient if she is having pain, she will anticipate and expect to experience it throughout the entire operation. At times the patient will complain of a dragging sensation or actual pain. Assure her that this is due to some other cause and not because the anesthesia has failed. If it is due to insufficient anesthesia either give more anesthesia or complete the operation under nitrous oxide. Another important factor is the amount of talking in an operating room. Absolute quiet is essential. The only one permitted to talk is the surgeon. Remarks such as, "stop that bleeder," must be avoided. It is also important to keep as few instruments on the patient as possible and have the assistants keep their elbows from resting on the patient. Make the patient as comfortable as possible.

The addition of adrenalin to the anesthetic solution is not recommended for the toxic thyroid group of patients. The administration of even small amounts of adrenalin causes the pulse rate to rise too rapidly and the patient has a feeling of impending danger. If the anesthesia wears off during the latter part of the operation nitrous oxide or some other form of inhalation anesthesia may be given.

Technique.—One per cent novocaine solution is employed for the superficial infiltration of the skin and the deeper tissue are infiltrated with ½ per cent solution. Using a No. 25 or 27-gauge needle the line of the proposed collar incision is infiltrated

with 1 per cent novocaine solution. This solution is injected intradermally and if done properly no pain or discomfort is experienced by the patient. Use new and sharp needles. Inject the solution as the needle enters the skin and the solution is introduced simultaneously with the forward progress of the needle. The bevel of

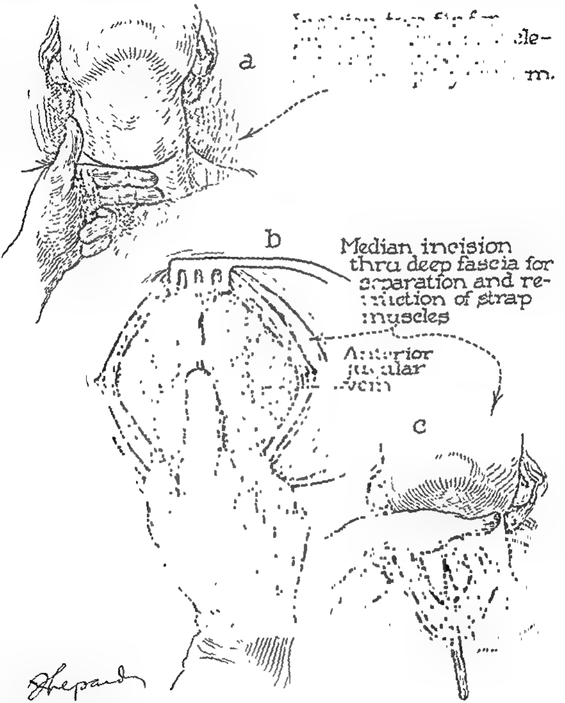

FIG. 77.—Thyroidectomy, (a) skin incision; (b) vertical incision through the deep fascia; (c) incision extended inferiorly.

the needle should face the skin. After the whole length of the skin incision has been infiltrated the deeper tissue are infiltrated with ½ per cent of novocaine. The area includes the supra-clavicular region, along the posterior border of the sternocleidomastoid muscles and area immediately underneath the line of the incision. For the deeper infiltration, 22 gauge 3 inches size needle is used. The cutaneous nerves of

the neck are now blocked with 10 cc. of 1 per cent novocaine. A wheal is made at the mid-point of the origin and insertion of the sternocleidomastoid muscle on its posterior border. This corresponds to about 1 centimeter above where the external jugular vein crosses the muscle. A 3 inch long needle is inserted through the wheal and carried forward until the resistance offered by the deep cervical fascia is encountered and the needle is then advanced through the fascia and the solution injected. This procedure will block the superficial sensory nerves of the neck and will give satisfactory anesthesia.

TECHNIQUE OF THYROIDECTOMY

The skin incision follows along the original line of infiltration. It is a modified "collar" incision extending from the right to the left external jugular veins, and about 1½ inches above the clavicle. The larger the gland the higher the incision is placed, (Fig. 77a). The incision is made through the skin, subcutaneous fat, and platysma muscle, exposing the superficial layer of the deep fascia and the anterior jugular veins. The upper and lower flaps of skin and subcutaneous layer of fat are reflected from the underlying deep fascia by sharp dissection. The upper flap is reflected as high as the hyoid bone in the midline and well above the superior poles on the lateral sides. The superficial layer of the deep fascia is now split in the midline (Fig. 77b) in the vertical plane. Bleeding points are ligated. The ribbon muscles are then split in the midline exposing the isthmus with its glistening capsule. The muscles are now retracted and separated from the underlying thyroid gland. The separation is done carefully with finger dissection. At about half-way down the lateral surface of the gland, the lateral thyroid vein is encountered, isolated and ligated. The separation is carried downward and laterally until the pulsating common carotid artery is felt. At this time, 5 to 10 cc. of 1 per cent novocaine solution is injected into the superior pole of the gland. The gland is now grasped with forceps and rotated over the trachea. With slight traction the superior pole comes into view. If the pole extends high in the neck, it may be necessary to detach the sterno-thyroid muscle from the thyroid cartilage. However, this is not usually necessary as it can be dislocated downward by gentle traction and freeing it with the handle of a scalpel from the esophagus or trachea. In this manner the pole is brought down so that the superior thyroid vessels become accessible. After the pole has been isolated a curved Kelly forceps is placed between it and the trachea and the pole is then completely separated from the trachea. Placing the index finger (Fig. 78a) underneath the pole with its tip at the inferior horn of the thyroid cartilage assures perfect isolation of the pole. After the forceps has made contact with [illegible] sterior blade of another curved Kelly [illegible] withdrawn, the new clamp closes over the superior pole as illustrated in Fig. 78b. Two more similar forceps are introduced and the superior pole is incised between the second and third clamps. The superior pole is now ligated in the groove of the most proximal clamp. Removal of the second clamp leaves a tit-like projection that serves to prevent slipping of the ligature. Instead of using forceps, the ligature may be passed around the pole with a needle.

The inferior pole is now grasped with thyroid forceps and elevated to expose the inferior thyroid veins. The vessels are grasped with forceps, cut and ligated. The lobe is now raised and rotated medially and straight Kelly forceps are applied into

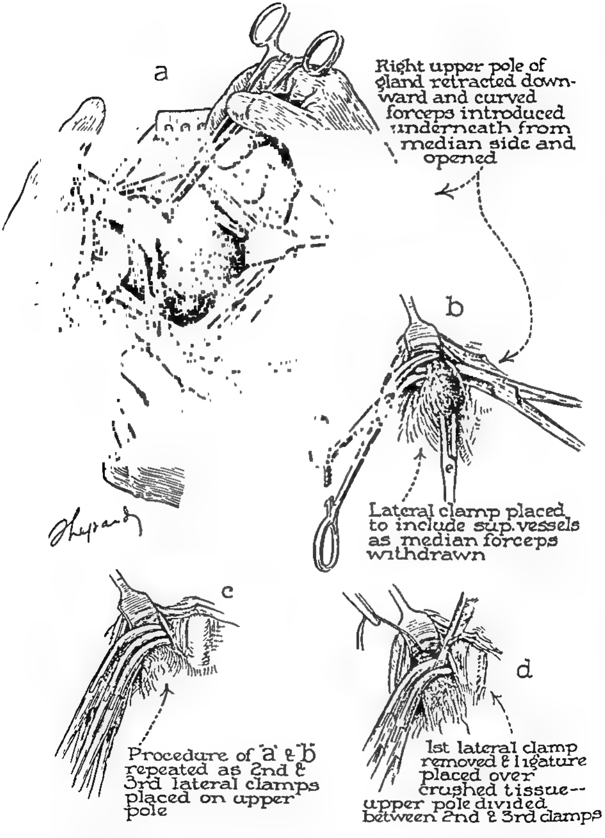

FIG. 78.—Ligation of the superior pole, (a) shows method of introducing a curved Kelly forceps to grasp the superior pole; (c, d) shows the method of ligating the stump of the superior pole.

the anatomical capsule from above downward. In this manner the lobe is resected in its lateral margin from above downward, leaving the posterior layer of the anatomical capsule and as much of thyroid gland as judgment dictates. The clamps are applied parallel to the gland to avoid injury to recurrent laryngeal nerve and the parathyroid bodies. The tips of the clamps are always in view. After the lateral aspect of the gland has been excised the lobe is separated from its mate by cutting the isthmus over a curved Kelly forceps placed between the gland and the trachea.

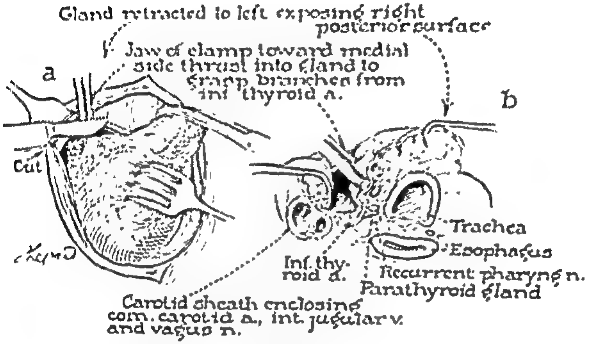

Fig. 79.—(a) The gland is retracted to the median line; (b) shows the scheme in the separation of the gland to avoid injuring the recurrent nerve.

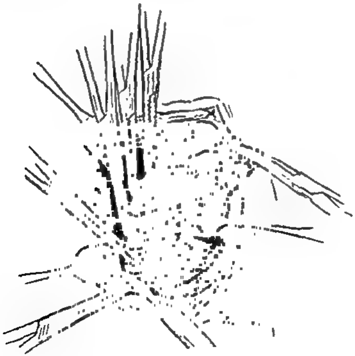

Fig. 80 —

The lobe is then excised from its medial aspect as illustrated in Fig. 83a. This completes the operation on one side and the removal of the other lobe is done in a similar manner. The remaining gland surface may be sutured if there is any bleeding.

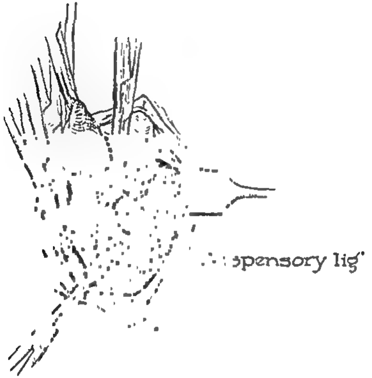

FIG. 81.—After the gland has been mobilized on its lateral aspect, it is then separated from its medial side by cutting the suspensory ligament between forceps.

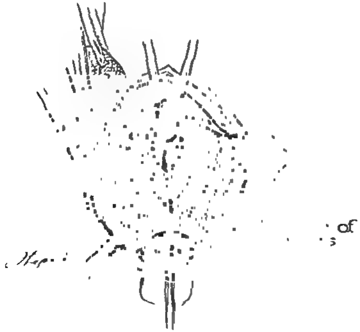

FIG. 82.—Method of dividing the isthmus.

If drainage is desired, a plain soft rubber strip cut in Y form is inserted in the beds formerly occupied by the thyroid lobes. The tail of the drain protrudes through a horizontal puncture wound made beneath the line of incision. The deep fascia is coapted with interrupted sutures. The platysma is likewise sutured with fine interrupted sutures and the skin is closed with a subcuticular stitch. A dry dressing is applied after a safety pin has been placed through the rubber drain.

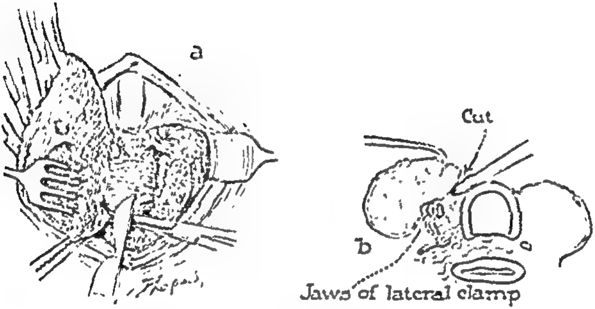

FIG. 83.—(a) The gland is excised from its medial aspect by grasping the gland with a forceps and cutting with scalpel; (b) shows the scheme of the excision.

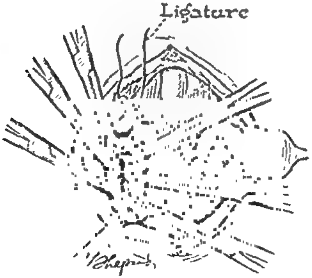

FIG. 84.—After the gland has been excised, the capsule may be sutured over the remaining portion of the gland.

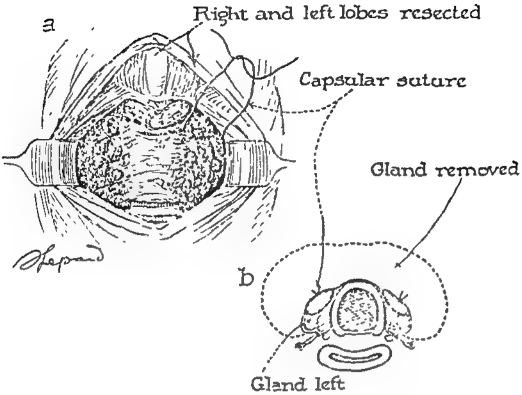

FIG. 85.—(a) Shows both lobes removed; (b) shows the scheme of the resection.

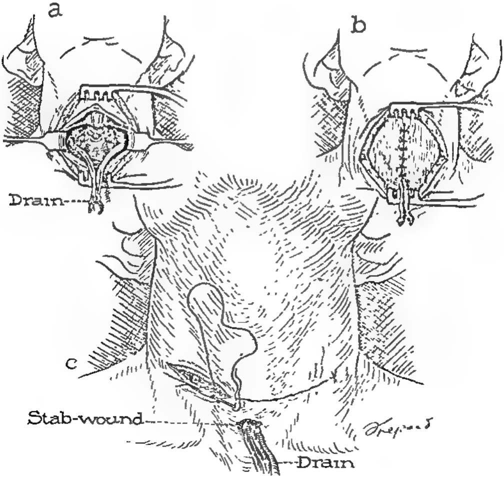

FIG. 86.—Thyroidectomy completed Shows the suturing of the deep fascia, the application of drainage, and the closure with a subcuticular stitch.

QUESTIONNAIRE

1. Discuss the embryology of the thyroid gland.
2. Define, thyroglossal cyst.
3. Describe the histological structure of the thyroid gland.
4. What is the role of iodine in thyroid function?
5. How much iodine is normally stored in the gland?
5. Discuss the role of iodine in thyroid diseases.
6. What is the principle action of thiouracil?
7. Give the contraindications of thiouracil therapy.
8. When is the use of thiouracil especially indicated?
9. How are the triangles of the neck formed?
10. What is the surgical significance of the midpoint of the sternocleidomastoid muscle?
11. What are the "ribbon" muscles?
12. Describe the cervical fascia of the neck.
13. How is the surgical capsule of the thyroid gland formed?
14. Describe the blood supply of the thyroid gland.
15. Describe the relationship of the recurrent laryngeal nerves to the thyroid gland.
16. Why is the nerves more apt to become injured when ligating the vessels of the inferior pole?
17. What are the advantages of local anesthesia in thyroidectomy?
18. Describe the technique of thyroidectomy.

CHAPTER 13

SURGERY OF THE NECK

TUMORS OF THE THYROID GLAND

By A. V. PARTIPILO and J. P. CONCANNON

OUR knowledge of the histopathology, treatment, and natural history of lesions of the thyroid gland has increased considerably in the last fifteen years. Many complex anatomical and histological problems have been solved. The scope of surgery has been extended. Radioactive iodine has opened a new avenue of approach to the diagnosis and treatment of thyroid carcinoma.

A sound knowledge of the pathology of thyroid tumors is essential in understanding the varied biological behavior patterns which modify both treatment and prognosis. One of the most difficult problems has been a classification of tumors of the thyroid gland. Some malignant lesions of the gland are difficult to classify pathologically. The histological distinction between a benign adenoma and a carcinoma may be extremely difficult since the usual criteria of malignancy do not always hold in thyroid neoplasia. An adenoma which was termed benign by microscopic examination may prove its malignant nature by metastasizing at a later date and the metastases themselves structurally may appear no different from the primary lesion which was termed benign.

Classification of Thyroid Tumors.—Many classifications of thyroid tumors have been described and the one presented here is a modification of an accepted classification. It well illustrates the differences in the natural history of the various tumors and indicates the appropriate methods of treatment as well as the prognosis. The first three groups of diseases of the thyroid gland listed in this classification, namely Graves's disease, adenomatous goiter and thyroiditis, are included in this discussion because they are often confused clinically and less frequently as tumors of the thyroid gland.

1. Primary Hyperplasia (Exophthalmic Goiter, Graves's Disease).
2. Adenomatous Goiter.
    a. Without secondary hyperplasia.
    b. With secondary hyperplasia and hyperthyroidism.
3. Thyroiditis.
    a. Acute and subacute thyroiditis.
    b Chronic thyroiditis.
    c. Struma lymphomatosa (Hashimoto's disease).
    d. Struma fibrosa (Riedel's disease).
4. Benign Tumors.
    a. Follicular adenoma
    b. Papillary adenoma (*see* Benign and Malignant Tumors, p. 184).
    c. Hürthle cell adenoma
5. Malignant Tumors.
    a. Follicular adenocarcinoma.

b. Papillary adenocarcinoma.
c. Alveolar carcinoma.
d. Hürthle cell type carcinoma.
e. Anaplastic carcinoma.
f. Others:
    (1) Squamous cell carcinoma due to metaplasia or arising from remnants of the thyroglossal duct.
    (2) Lymphoblastoma.
    (3) Metastatic carcinoma.
    (4) Mixed tumors.

Primary Hyperplasia.—Graves's disease is mentioned only as of passing interest since the classical symptoms and eye signs of this disease make the diagnosis rather obvious and distinguish it from other enlargements of the thyroid gland. According to Meissner, the picture of the severe hyperplastic epithelium of Graves's disease in a gland that has been previously damaged, may give rise to considerable difficulty in making a pathological diagnosis. He further points out that in some cases of Graves's disease there is extension of hyperplastic epithelium into the adjacent fibrous tissue, and even into adjacent skeletal muscle. Such extension, according to Meissner, must not be considered as evidence of carcinoma.

Adenomatous Goiter.—Adenomatous goiter with or without secondary hyperplasia is seen most frequently in areas of endemic thyroid diseases. The nodular nontoxic goiter without secondary hyperplasia may manifest itself as a single, firm and prominent tumor which may be difficult to differentiate from carcinoma. If the tumor is soft or there are signs of hyperplasia the chances of malignancy are rather remote. Again, clinically it is almost impossible to say that a single solitary nodule in the thyroid gland is an adenomatous goiter or a true adenoma. In endemic areas the great majority of these are most likely to be diagnosed by the pathologist as adenomatous goiter. With the large adenomatous goiter producing compression symptoms and having multiple nodules, the diagnosis is not difficult. The treatment of the nontoxic adenomatous goiter is subtotal lobectomy. The treatment of the toxic goiter is discussed in Chapter 12.

Thyroiditis.—The various pathologic diseases under the heading of thyroiditis are included in this discussion because from the history and physical findings it is difficult, if not impossible, to differentiate these enlargements from true neoplasms of the thyroid gland. Most of these respond to medical treatment and surgery is indicated only for compression symptoms. It is therefore desirable that a clinical diagnosis be made in order to avoid unnecessary surgery.

An important diagnostic feature of a patient with subacute thyroiditis is the history of an upper respiratory infection, or repeated infections of the throat one or two months preceding the onset of the enlargement and tenderness in the neck. The enlargement in the neck is usually diffuse, however, it may be localized. According to Crile and Dempsey, there is characteristically a greatly elevated sedimentation rate and an absence of radioactive iodine uptake by the gland. Crile suggests that needle biopsy be done with a Silverman needle to help in the diagnosis. If confirmed the patient is treated with antibiotics and cortisone. If the diagnosis is made by frozen section during surgery, it is not necessary to remove the gland.

Another thyroid enlargement which defies clinical diagnosis is Hashimoto's disease. The disease should be suspected whenever a middle-aged woman with symptoms of hypothyroidism has an enlarged and firm thyroid gland. Although

the etiology is unknown, it is probably associated with hormonal disturbance since it occurs in 90 per cent of cases in the female during their menopausal stage. The significant features of this disease are the diffuse enlargement of the gland over a year period and progressive hypothyroidism. In the later stages of the disease the growth causes pressure on the trachea. The differential diagnosis includes tumor, diffuse hyperplasia, and chronic thyroiditis. During surgery the gland is found to be uniformly enlarged, rather firm but not stony hard, and easily separated from the surrounding structures. Because of its firm character it can be easily mistaken for carcinoma, however its diffuse enlargement and non attachments are evidences against malignancy. Biopsy will confirm the diagnosis without any difficulty. The microscopic picture shows a diffuseness and uniformity of acidophilia of the epithelium and round cell infiltration with definite germinal centers. For this reason this disease is often referred to as a lymphomatosa. Lymphocytic infiltration is also seen in chronic thyroiditis, Graves' disease, adenomatous goiter and in various other tumors. However, the finding of germinal centers is specific in Hashimoto's disease. If the diagnosis is made by frozen section, resection of the gland is not indicated since its removal would only hasten the hypothyroidism. The treatment of this disease is with dried thyroid extract. Surgery is indicated only for the relief of compression symptoms.

The last of the diseases under the classification of thyroiditis is Riedel's struma. This disease involves localized areas of the gland. It is stony hard and is fixed to its surrounding structures and for this reason it is often mistaken for carcinoma. It is recognized microscopically by the marked fibrous replacement of the involved portion of the gland. It is not preceded by inflammatory reaction. The patient's chief complaint is from pressure on the trachea. During surgery it may be difficult to differentiate this disease from carcinoma, however the absence of enlarged lymph nodes will indicate its true nature. The treatment is partial lobectomy.

Benign and Malignant Tumors.—Primary carcinoma of the thyroid gland represents a heterogenous group of tumors of widely different histological patterns which makes it difficult to correlate these different patterns with the clinical course of each type. The less malignant types offer equally confusing and troublesome problems. Some malignant lesions are also difficult to classify pathologically. The histological distinction between a benign adenoma and a carcinoma may be extremely difficult since the usual criteria of malignancy do not always hold in thyroid neoplasia. Surgeons have had the experience of removing an adenoma which was termed benign by microscopic examination only to have its malignancy demonstrated by metastasizing at a later date with these metastases proving to be structurally no different from the primary lesion. It is important to understand that the pathologist will be unwilling to make a diagnosis of approximately half of the specimens submitted for frozen section studies because time would not permit serial sections to firmly establish the pathological diagnosis. In these cases lobectomy and the removal of the isthmus is done. A more thorough procedure is subsequently done if full pathological examination reveals malignancy. Certainly, evidence of blood vessel and capsular invasion or the finding of metastases despite an otherwise benign appearance microscopically, places an "adenoma" into the category of malignant neoplasia. The presence of papillary structure in a thyroid neoplasm is likewise strong evidence of malignancy, especially if this occurs in a younger individual. Not all thyroid cancers are so difficult to evaluate, since there can be no doubt about the malignant nature of a thyroid neoplasm when the tumor has anaplastic

features or when there is evidence of invasion or metastases. Although a tumor of only one type of thyroid carcinoma may be seen, it is not unusual to see tumors with components of several different types, as well as varying degrees of differentiation within the same tumor if multiple sections are studied (Figs. 87 and 88.) A tumor which is primarily a well differentiated colloid containing adenocarcinoma may have

FIG. 87

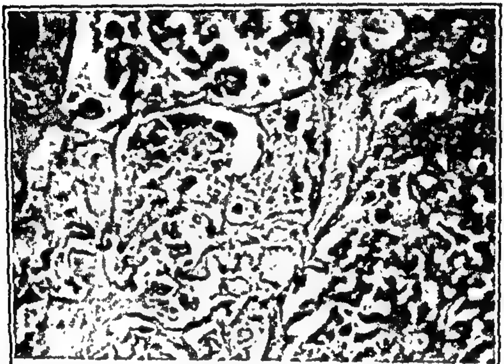

FIG. 88

FIG. 87.—Papillary adenocarcinoma of the thyroid with areas of hemorrhage. In some areas nests of undifferentiated cells are seen (H. E. × 100).

FIG. 88.—Same tumor as in Figure 87. Solid anaplastic carcinoma with well defined clusters of undifferentiated cells without follicle formation. In the middle of the picture are cells of alveolar adenocarcinoma (H. E. × 100).

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FIG. 87

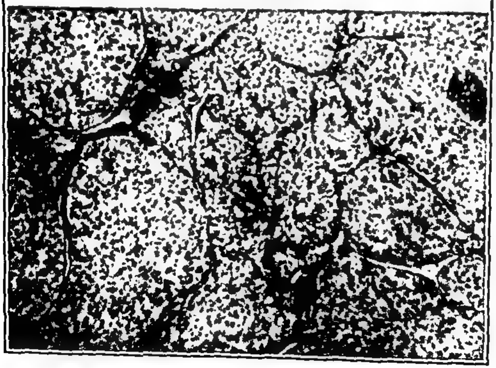

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FIG. 87.—Papillary adenocarcinoma of the thyroid with areas of hemorrhage. In some areas nests of undifferentiated cells are seen (H. E. × 100).

FIG. 88.—Same tumor as in Figure 87. Solid anaplastic carcinoma with well defined clusters of undifferentiated cells without follicle formation. In the middle of the picture are cells of alveolar adenocarcinoma (H. E. × 100).

elements of papillary adenocarcinoma as well as other areas where anaplastic carcinoma may be seen.

Another problem which continues to plague both the surgeon and the pathologist is whether an adenoma can turn into cancer, or whether the so-called adenoma is cancerous from the very beginning. This is difficult to prove because once an adenoma has been removed, it would be presumptuous to postulate as to what would have resulted if it had not been removed. On the other hand, it is not unusual to see patients who have had solitary nodules for many years without evidence of growth and finally start to grow and after removal these tumors prove to be malignant. An adenoma may undergo any one of the following courses: (1) First of all, it may remain dormant even throughout the life of an individual without evidence of any changes; (2) secondly, it may slowly increase in size as a result of retrogressive changes such as hemorrhage, fibrosis, calcification, round cell infiltration or cyst formation; (3) thirdly, hyperplasia may occur giving rise to hyperthyroidism and the so-called toxic adenoma; (4) Lastly, it may be transformed into a carcinoma. It has been variously estimated that up to ten per cent of adenomas turn into cancer.

Follicular Adenocarcinoma.—Follicular adenocarcinoma of the thyroid is a well differentiated neoplasm closely resembling normal thyroid tissue. Blood vessel invasion or metastatic disease may be the only indication of the malignant nature of an adenomatous tumor which otherwise appears to be benign. However, if multiple sections are examined, blood vessel invasion will be found in most instances of follicular adenocarcinoma (Fig. 89). It was this type of tumor which, when found in bone, was referred to by Conheim as "benign metastasizing goiter." This concept is now only of historical interest as Berard and Dunne, Joll, and others have shown that adequate examination of these metastasizing thyroid tumors will always show evidence of carcinoma. The term "adenoma with blood vessel invasion" has also been applied to this type of tumor and likewise this term appears to be a misnomer since the tumor described is really a well differentiated thyroid cancer.

Metastases when present are nearly always due to hematogenous spread. Although tumor dissemination through the lymphatic channels is relatively rare, cervical glandular metastases may occur occasionally if the tumor capsule is invaded.

Clinically, a follicular adenocarcinoma is a slow growing tumor which may have existed for years before causing local signs or producing metastasis. Very often the tumor may have been discovered accidentally by the patient or by a doctor during a routine physical examination. Frequently, surgery may have been done as a prophylactic measure without the clinical suspicion of its malignant nature until after the pathological examination. On the other hand, surgery may have been done because of local pain or enlargement of the nodule with a preoperative diagnosis of possible malignancy.

The recurrence rate and the incidence of local or remote metastases is low even when microscopic evidence of blood vessel invasion is found. The prognosis is good and in the many series reported, there have been few deaths due to this type of tumor.

The treatment of follicular adenocarcinoma is total lobectomy of the involved lobe. This is sufficient to produce cure. This type of tumor because of its well differentiated cellular structure is relatively radio-resistant. It will be only in those few patients who have distant metastases when first seen, that radiation therapy will be helpful. External radiation therapy has little to offer, but therapy with radioactive iodine may be of value.

During surgery the operative field should be thoroughly explored for venous involvement. If the thyroid veins appear to be involved they should be removed in continuity with the thyroid lobe. In the more advanced stage the internal jugular vein should be ligated above the superior thyroid vein and below the inferior thyroid vein and this should be done before starting the dissection of the involved lobe. This segment of the jugular vein is then excised with the thyroid lobe. If

FIG. 89

FIG. 90

FIG. 89.—Follicular adenocarcinoma of the thyroid showing evidence of blood vessel invasion (H. E. × 430).

FIG. 90.—Papillary adenocarcinoma of the thyroid (H. E × 100).

elements of papillary adenocarcinoma as well as other areas where anaplastic carcinoma may be seen.

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Fig. 89

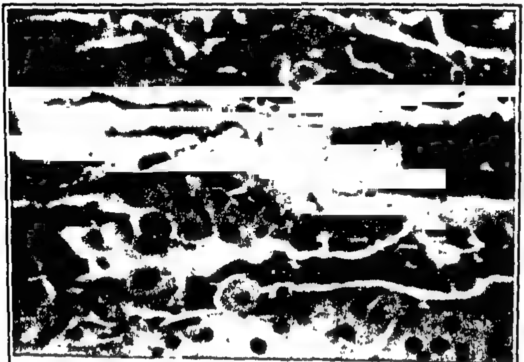

Fig. 90

Fig. 89.—Follicular adenocarcinoma of the thyroid showing evidence of blood vessel invasion (H. E. × 430).

Fig. 90.—Papillary adenocarcinoma of the thyroid (H. E. × 100).

the tumor appears inoperable, palliative procedures are instituted with the objective of restoring and maintaining a free airway. This may require the removal of as much of the malignant tissue as possible in an effort to relieve pressure on the trachea. Tracheotomy should be considered when extensive dissection is done especially when a large mass is left behind and radiation therapy is contemplated.

Papillary Adenocarcinoma.—Papillary adenocarcinoma is primarily a localized, encapsulated, slow growing neoplasm with characteristic papillary morphology. The lesion may be present for a long period of time before evident changes are clinically observed. As with malignant adenomas, there is some difference of opinion as to whether papillary adenocarcinomas arise from pre-existing papillary cystadenomas or whether they are malignant from the start. Many pathologists feel that it is impossible to state that any given papillary neoplasm is benign. It is now generally accepted that the presence of papillary structure in a thyroid neoplasm indicates carcinoma (Fig. 90). Clinically, there is considerable evidence to substantiate this view. In a series of patients analyzed at the Lahey Clinic, it was shown that the duration of the neoplastic disease prior to surgery, average survival and five-year survival rates following treatment, as well as the percentage dead of this disease was approximately the same for the patients in the papillary cystadenoma group as for the patients classified as having papillary adenocarcinoma. Next to follicular carcinoma, papillary adenocarcinoma has the best prognosis of all malignant tumors of the thyroid gland.

These papillary tumors tend to occur in a younger age group than in those patients with alveolar adenocarcinoma. Although papillary adenocarcinoma occurs in adults, it is the characteristic thyroid cancer of youth and childhood. The primary tumor is often distinguished by its extremely small rather than large size and is frequently present as a palpable nodule in the thyroid for a period of years before surgical treatment is sought. They tend to metastasize to regional lymph nodes over a period of years. It was these slow growing metastases that were referred to in the past as "lateral aberrant thyroid tumors." Today, it is generally accepted that these lateral aberrant thyroid tumors are nearly always metastatic extensions to the deep cervical lymph nodes from a primary carcinoma of the homolateral lobe of the thyroid gland (King and Pemberton). Willis states that if a hemi-thyroidectomy is done when such tissue is found, a small carcinoma in the homolateral lobe is always found.

When the patient is first seen, the primary tumor may be either a small nodule in the thyroid gland or a large mass. Palpable unilateral lymph node involvement is present in more than half the patients at this time and a lesser number will have bilateral lymphadenopathy. The cervical lymph nodes secondary to papillary adenocarcinoma of the thyroid may achieve great bulk. In some patients metastatic disease in cervical nodes may be the only palpable indication of neoplastic disease, and it is only on careful examination of the removed homolateral lobe that the primary tumor can be detected. Blood vessel dissemination may occur with involvement of lungs and bone. A relatively small number of patients have distant metastases when first seen.

Papillary adenocarcinomas have been considered as low grade tumors because of the prolonged clinical course of the disease and it is not unusual for distant metastases to be present for many years before death occurs. However, when the patients are followed over a considerable period of time, the mortality rate due to the disease continues to increase. The most frequent cause of death is local extension

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3. The so-called "lateral aberrant thyroid tumor" must also be considered as a metastatic invasion of the deep cervical lymph nodes even though a palpable tumor cannot be detected in the homolateral gland. In this case a bilateral thyroidectomy combined with radical neck dissection should be done. Metastatic lymph nodes are usually freely movable, remain localized, and can be easily removed without any tendency toward recurrence.

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the tumor appears inoperable, palliative procedures are instituted with the objective of restoring and maintaining a free airway. This may require the removal of as much of the malignant tissue as possible in an effort to relieve pressure on the trachea. Tracheotomy should be considered when extensive dissection is done especially when a large mass is left behind and radiation therapy is contemplated.

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Some tumors contain clear cells which resemble those of the clear cell adenocarcinoma of the kidney. If multiple sections of the specimen are examined other structural variants of thyroid cancer may be discovered. These tumors, when malignant, frequently metastasize to cervical lymph nodes and blood borne metastases are not infrequent. The clinical course is prolonged but appearance of lymph node metastases will ultimately occur in 15 per cent, bone metastases in approx-

Fig. 91

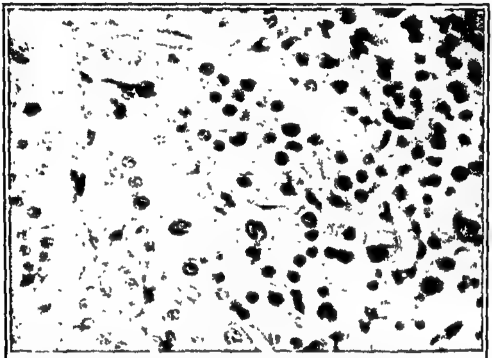

Fig. 92

Fig. 91.—Hurthle cell carcinoma of the thyroid. Cells irregularly sized but all large with homogeneous vacuolized eosinophilic cytoplasm (H. E × 430).

Fig. 92.—Anaplastic carcinoma of the thyroid (H. E. × 100)

nately 15 per cent and lung metastases in approximately half the patients. Only bout one-third of the patients with this type of tumor will be alive and well without vidence of disease five years after surgical treatment. External radiation therapy justified only if there is evidence of residual tumor following an adequate surgical rocedure.

Alveolar Adenocarcinoma. The histologic appearance of alveolar adenocarcinoma may vary considerably. At one end of the scale there may be numerous cinar structures, with cells only slightly less differentiated than those of follicular denocarcinoma. At the other end of the scale there is a transition toward anaplasia ith the retention of some acinar structures permitting the classification of neolasm as an alveolar adenocarcinoma. Between these two extremes will be found umors of varying degrees of differentiation.

The less well differentiated alveolar adenocarcinomas tend to spread locally ithin the thyroid gland and become fixed to surrounding structures by local inasion. Cervical nodes become involved following invasion of the capsule and djoining structures. Metastases most frequently occur by blood vessel route with nvolvement of lungs, bone, and brain. Occasionally, symptoms produced by disant metastases may be the first indication of malignant disease and even more atients will have distant metastases when first seen.

The prognosis for patients with alveolar adenocarcinoma of the thyroid is much worse than for the patients in the previously discussed groups. The response of alveolar adenocarcinoma to radiation therapy is variable. The highly differentiated alveolar adenocarcinoma tend to be radio-resistant. The less differentiated alveolar adenocarcinomas are more sensitive with a response not unlike the anaplastic carcinomas. In these patients postoperative radiation is of value in diminishing the local recurrence rate.

Some alveolar adenocarcinomas will have sufficient iodine avidity to warrant therapy with radio-iodine. Other tumors of this type even after thyroid ablation will show no evidence of concentrating I^{131}. If testing after abaltion of the thyroid shows no iodine concentrating tissue, then the patient should have the benefit of local *x*-ray therapy. Small portals 6 × 8 cm. in size are arranged about the neck and a tumor dose of 4500 to 5000 roentgens is given.

Anaplastic Carcinoma.—Anaplastic carcinomas tend to occur in patients of an older age group than the alveolar carcinomas. Histologically, the tumor may be pleomorphic with round, polyhedral, and spindle cell shaped cells. The nuclei tend to be bizarre, hyperchromatic and have numerous mitotic figures. Multinucleated giant cells are a common feature of the anaplastic carcinoma of the thyroid (Fig. 92).

The anaplastic carcinomas are the most malignant of the thyroid tumors. The history is usually of a short duration and rarely is there a history of a previously existing adenoma. There may only be a history of a few weeks growth in the neck when the patient is first seen. The spread of the neoplasm is diffusely throughout the gland and early invasion of adjoining anatomical structures occurs. The tumor may rapidly encircle, compress and invade the esophagus and trachea, causing esophageal and tracheal obstruction. Local growth may involve the recurrent laryngeal nerve causing laryngeal palsy and even the vagus nerve may be involved. A Horner's syndrome will occur if there is involvement of the cervical sympathetics. Since the anaplastic carcinoma is so frequently seen at the base of the neck it is not unusual to see the sternum and clavicle invaded by the neoplasm. In a relatively short period of time there may be a bulky mass fixed to many of the adjoining

structures in the neck. Metastatic spread is early by hematogenous dissemination as well as lymph node spread. Frequently, spread of the disease to the upper mediastinum causes superior vena caval obstruction. More than half of the patients have lymph node metastases when first seen and distant metastases are present in a lesser number. Death may be due to rapid local invasion before the development of metastases, due to metastatic involvement alone, or due to the combination of both processes.

The majority of patients, when first seen, have massive local involvement beyond the scope of operability but fortunately many of these tumors are very radiosensitive. These patients are often admitted to the hospital with compression of the trachea for which they should be treated as external radiation therapy emergencies. Many of these patients have alleviation of symptoms following the first few x-ray treatments and not infrequently great bulky masses disappear with great rapidity. Despite extreme radio-sensitivity, distant metastases or early local recurrence often prevents cure.

In view of the extremely malignant nature of these tumors, it is quite remarkable that there should be any survivors five years after treatment. Windeyer cites a series of 28 patients with anaplastic carcinoma of the thyroid and of these, 8 were living five years later. Hare reported 6 patients alive out of 30 who had x-ray therapy for anaplastic carcinoma. Local recurrence and distant metastases following radiation therapy keep the five year survival figures relatively low.

In the more diffusely infiltrating anaplastic tumors, it is usual to use large field therapy, *including large enough portals to cover the upper mediastinum as well as* the cervical area. The field sizes used range from 10×15 cm. to 10×20 cm. If there is early reduction in tumor size, it may be advantageous to reduce the portal size and raise the tumor dose to higher levels.

Clinical Staging.—Although pathological staging is of utmost importance in achieving an understanding of the disease, a clinical staging is of equal importance in determining prognosis and appropriate methods of treatment. Folke Jacobson has presented a clinical staging of thyroid carcinoma similar to classifications for carcinomas primary in other sites.

Stage I. The primary tumor is mobile or only slightly fixed to adjacent structures.

Stage II. The primary tumor is mobile or only slightly fixed to adjacent structures. Mobile lymph node metastases on one side of the neck.

Stage III. The primary tumor is extensively fixed to adjacent structures. Cases with bilateral or fixed lymph node metastases in the neck.

Stage IV. Distant metastases are present.

Prognosis must be based not only on histological type of the tumor but also on the clinical staging of the disease. Naturally, because of the rapid growth of anaplastic carcinomas and the less well differentiated alveolar adenocarcinomas there will be a greater proportion of these tumors in the advanced stages while the more differentiated tumors will tend to be in the earlier stages. Again, a clinical staging *is of importance in emphasizing the need for a clinical suspicion of malignancy* when a nodular lesion of the thyroid is discovered and this is particularly so when this is found in an individual below the age of thirty. The incidence of carcinoma in nodular goiter is said to vary from 4 to 18 per cent and it has also been observed that there is a much higher incidence of carcinoma in the solitary nodular goiters

9. How is Riedel's struma recognized microscopically?
10. How would you treat it?
11. Discuss the relationship between benign and malignant tumors of the thyroid gland.
12. What gross findings places an "adenoma" into the category of a malignant neoplasm?
13. Does the finding of papillary structure have any significance?
14. Discuss the various changes that an adenoma may undergo?
15. Describe the microscopic picture of a follicular adenocarcinoma.
16. What is meant by "Conheim's benign metastasizing goiter"?
17. How do follicular adenocarcinoma spread?
18. What is the prognosis of papillary and follicular adenocarcinoma?
19. What is the treatment of follicular adenocarcinoma?
20. Is radiation therapy of value for these tumors?
21. Discuss "lateral aberrant thyroid tumors."
22. How would you manage a case of papillary adenocarcinoma?
23. Discuss Hürthle cell tumors.
24. What is the prognosis for alveolar adenocarcinoma?
25. How would you treat it?
26. How would you treat anaplastic carcinomas?
27. Give the clinical staging of thyroid carcinomas.
28. Discuss the general principles in the treatment of carcinomas of the thyroid gland.

Chapter 11

SURGERY OF THE NECK—RADIOACTIVE IODINE IN CANCER OF THE THYROID

By J. P. Concannon and Gerald E. Nora

Radioactive Iodine has been used in the treatment of thyroid carcinoma since 1942. Numerous reports have been published concerning therapy with this agent and after twelve years it is becoming possible to make some preliminary assessment of its value. This form of treatment has been found effective only in a small proportion of patients with carcinoma of the thyroid, but this in group there have been some who have shown evidence of remarkable regression of disease.

Although the selection of cases of thyroid carcinoma for radio-iodine treatment is relatively simple in principle, there are a number of difficulties encountered in practice. Simply, those patients with thyroid cancers which acquire, concentrate, and hold the radioactive Iodine are the ones who should receive this form of treatment.

To warrant therapy with radio-iodine it is first necessary to *demonstrate* that neoplastic tissue acquires Iodine, and holds it for a reasonable period of time. There are numerous ways in which acquisition of I^{131} by neoplastic tissues can be determined and measured. The patient may be given a diagnostic quantity of radio-iodine a few days prior to surgery and the quantity of I^{131} measured in microcuries per milligram of surgically removed neoplastic tissue. Since a certain percentage of the activity determined will be due to inorganic and organic $Iodine^{131}$ in blood, the figure obtained can be corrected by subtracting the microcuries of radio-iodine per milligram of removed muscle. This is a very satisfactory procedure when the carcinomatous tissue studied in this manner, is taken from an area remote from the thyroid. The test may be misleading if the nodule is taken from the thyroid, since the activity determined may be due to I^{131} in included normal thyroid tissue.

An autoradiograph is more reliable in obtaining evidence of uptake in neoplastic thyroid tissue. A quantity of I^{131} is given to the patient a few days before the surgical procedure. Strips of film are then exposed to mounted microscopic sections of the removed thyroid neoplasm for various periods of time, and if sufficient radio-iodine is present, the film will be exposed. When these preparations are studied under the microscope it can be determined how homogeneously the I^{131} and thus the radiation is spread through the tumor.

Needless to say, there are limitations to both these methods of examination. They give information only regarding the tissue removed and give no information concerning the function of other known metastases, nor do they demonstrate clinically unsuspected ones. The autoradiograph is frequently negative in the presence of normal functioning thyroid tissue and usually only following ablation of the normal thyroid, can a positive autoradiogram of tumor tissue be obtained. It is also difficult to follow the response of the tumor to radio-iodine therapy by radio-

Most methods and techniques for total body scanning in general use today have limitations. The rounded, commercially available collimators "see" only relatively small areas. It is usual in doing a body scan with this type of collimator to scan along three or four parallel lines, determining the counting rate at 5-centimeter intervals along these lines. This is a tedious process and may require the services of two technicians working for two hours to complete a single body scan. Automatic scanning devices utilizing Geiger counters and scintillation counters to shorten the time of total body scanning as well as to improve representation of radioactive distributions have been described (Fig. 94).

The question arises when should this total body scan be done? There is some disagreement among authorities as to the optimal time after ingestion of radio-iodine for external scanning. Some believe that the patient should be scanned forty-eight hours later so that the metastases of anaplastic thyroid carcinomas which acquire but rapidly discharge radio-iodine will be detected. Others feel that total body scanning at seventy-two to ninety-six hours, when the total body background is low, will result in the detection of metastases from well differentiated thyroid carcinomas, even if only small quantities of radio-iodine are concentrated in thyroid cancer tissue. Our laboratories have followed Pochin's example of scanning the body daily as long as there is appreciable evidence of I^{131} retention within the body. This is a tedious routine but if functioning metastases are to be discovered, perseverance and thoroughness are necessary.

It is not just sufficient to scan the body in search of metastases. A permanent record of the distribution of radio-iodine within the body is desirable so that comparisons may be made at a later date when tracer and therapeutic quantities are given. This record will also make the examiner familiar with the areas of the body in which there is a normally higher concentration of radio-iodine. Unless the examiner is familiar with the patterns of normal iodine concentration within the body at various time intervals it is possible that a normal concentration of radio-iodine in the stomach, liver, bladder, kidney, and perhaps salivary glands will be mistaken for a functioning metastasis. (Fig. 95)

Iodine studies with complete body scan utilizing external scanning devices, such as scintillation counters, have frequently been successful in demonstrating metastatic deposits previously unsuspected by routine clinical methods of examination. In a personally reported series, five metastatic deposits in bone, four which were nonproductive of symptoms and unsuspected clinically, were detected by the use of a scanning device. The scan demonstrated uptake in the skull of a patient who had complained of headaches for a period of several months. Despite the very definite evidence of uptake in the region of the mastoid, careful examination of the radiograph showed no evidence of bony involvement. It was not until three months later that radiographic examination of the skull demonstrated an osteolytic lesion in the mastoid. The scan on another occasion demonstrated a lesion in the pubis which had not been detected in the roentgenogram, partly because of confusing overlying gas shadows, since later review of the films revealed the hidden osteolytic lesion in the pubis.

The detection of these metastases is of utmost importance, since if the uptake is adequate, therapy with radio-iodine is warranted. If the percentage uptake is adequate for diagnosis but not for therapy then it may be possible to enhance the uptake by thyroid ablation if the thyroid is present, or by courses of propylthiouracil or thyroid stimulating hormone if the thyroid has already been ablated.

What types of thyroid tumors can be expected to acquire, concentrate and retain radio-iodine? Naturally it is those tumors which most resemble normal thyroid tissue, the follicular adenocarcinomas and the well differentiated alveolar adenocarcinomas. Histology alone, however, does not preclude the possibility of a thyroid neoplasm acquiring radio-iodine since other histological type tumors may show evidence of Iodine avidity.

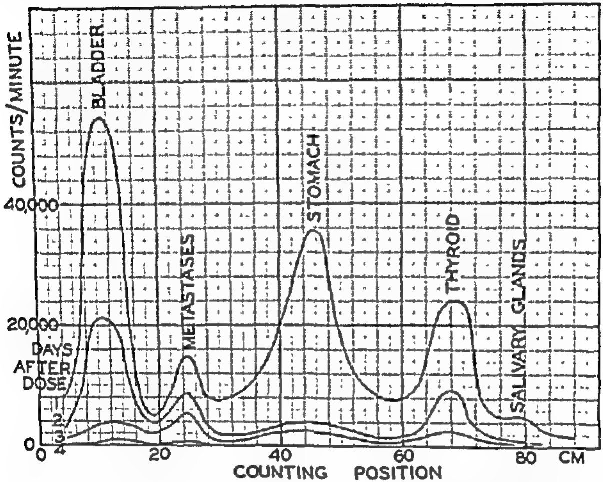

FIG. 95.—Record of counts detected by scintillation counter over various regions of the body on successive days. The record indicates the position of the functioning metastases.

In a personally reported series, 48 patients with thyroid cancer were studied. Table 7 indicates the histological classification of the tumors in the 48 patients studied and in the 13 patients who had enough uptake of I^{131} in these tumors to warrant therapy.

TABLE 7

	Total Number	*Number, No Uptake*	*Number, Uptake*
Adenocarcinoma	14	10	4
Papillary Adenocarcinoma	12	10	2
[illegible]	9	9	0
[illegible]	4	2	2
[illegible]	4	2	2
[illegible]	5	2	[illegible]
TOTAL	48	35	13

From inspection of this table it becomes apparent that no hard and fast rules can be laid down about which type of tumor will acquire radio-iodine. There were

10 patients who had tumors which might have been expected to acquire radio-iodine^{131} but which did not. Two patients had radio-iodine concentration in tumor tissue despite a diagnosis of papillary adenocarcinoma, as did 2 out of 4 patients who had Hürthle cell carcinomas. Even an anaplastic carcinoma may pick up iodine in sufficient quantities to warrant treatment with this agent. One of the patients listed in Table 7 despite a transition of the tumor type from a well differentiated adenocarcinoma to an anaplastic carcinoma, continued to exhibit some uptake in anaplastic tumor tissue in neck and thorax. The literature abounds with reports of patients with histologically unfavorable tumors which acquired radio-iodine. This histology alone therefore should not be a deterrent to the performance of iodine^{131} uptake studies.

The correlation of the clinical evolution and the clinical staging of the disease in relationship to the histology is most helpful in determining which patients should be studied with radio-iodine. It is obvious that there is not time to proceed with radio-iodine studies in a patient who has an anaplastic carcinoma growing wildly in the neck, producing tracheal and esophageal obstruction or an upper mediastinal syndrome. These patients should be treated as radiation therapy emergencies and given the benefit of immediate external radiation therapy to the local tumor mass. After an intensive course of such therapy, diagnostic quantities of I^{131} may be given to see whether remote metastases which may be present will show evidence of radio-iodine uptake. It is unusual for these anaplastic lesions, even if they show evidence of I^{131} uptake, to hold the radio-iodine long enough to have a beneficial effect on tumor tissue. If tracer studies now show no evidence of iodine retention by metastatic lesions from an anaplastic carcinoma then the metastases should be treated by external radiation therapy.

The patients with follicular adenocarcinoma in whom all evidence of disease has been removed surgically do not require Iodine^{131} studies. Tracer quantities may be given to search for distant unsuspected metastases but if functioning normal thyroid tissue is still in the neck, there is little likelihood of these unknown lesions acquiring a detectable quantity of radio-iodine. Radio-iodine ablation of the gland may be done if local recurrence or remote metastases occur at a later date.

Radio-iodine studies should be reserved for all patients with evidence of residual local disease following adequate surgical procedures, all patients with known distant metastases, all patients with papillary adenocarcinomas and alveolar adenocarcinomas and those patients with the more slowly growing anaplastic carcinomas which do not represent an immediate threat to life.

What is the usual plan of attack in patients with thyroid cancer? Pochin has outlined very nicely a plan to be used when thyroid carcinoma is suspected.

"First, biopsy.

"Secondly, radical excision if possible. If attempted and found to be impossible and biopsy has shown a differentiated tumor, the excision should include as much thyroid tissue as practicable.

"Inoperable tumors that are undifferentiated and appear likely to be radio-sensitive should be treated by radio therapy if sufficiently localized and unless they are shown to take up radio-iodine.

"With inoperable but well differentiated tumors and certainly for all cases with colloid filled follicles throughout the tumor, the thyroid should be ablated by total thyroidectomy if possible and otherwise by radio-iodine."

After thyroid ablation, radio-iodine treatment should be instituted if uptake

can be detected or induced in a tumor or simply if profile counting reveals any abnormal site of radio-iodine retention and continued until no such site remains, if this can be achieved without the development of radiation anemia.

It will be observed that much of the selection of cases can be done without the use of radio-iodine tests which are rarely profitable or essential before thyroid ablation. For most patients, therefore, no information is lost and valuable time may be gained if a biopsy is performed on the presumptive diagnosis of thyroid carcinoma and is followed by total thyroidectomy if the tumor proves to be highly differentiated. Radio-iodine is only then required after ablation, or for ablation, if thyroidectomy is impracticable."

Surgical ablation of the normal thyroid gland is preferred because of the rapidity with which iodine testing can follow this procedure. The patient has usually recovered from the operation sufficiently in ten to fourteen days so that iodine testing can be done at this time. When the thyroid is ablated by $Iodine^{131}$, the desired radiation effect does not become apparent for six to eight weeks after the administration of the therapeutic quantity of radio-iodine. Furthermore, there is some danger that the ablation dose of radio-iodine may reduce the concentrating power of the metastases. Radiation ablation should be reserved for those few patients who are inoperable because of general physical condition or for those patients who refuse any surgical procedures other than biopsy. To produce complete radiation ablation of the thyroid it is necessary to give quantities of radio-iodine in the range of 50 to 100 millicuries of I^{131}.

Following ablation of the thyroid whether by surgery or radiation therapy, a total body scan, after the patient has been given a diagnostic dose of radio-iodine, may now show evidence of enough uptake in neoplastic tissue to permit therapy with this radioactive agent. In a great proportion of patients, however, there again will be no evidence of Iodine concentration in cancer tissue although a few may have evidence of weak concentration insufficient to produce a desirable radiation effect in the neoplastic tissue. Certain chemical agents may be utilized to induce uptake or to increase the uptake in those areas where previously a slight concentration of radio-iodine was detected. It is the usual experience though that in those patients who show no evidence of radio-iodine uptake in neoplastic tissue following thyroid ablation, that the repeated use of propylthiouracil or thyroid stimulating hormone usually will not cause the neoplastic tissue to become iodine avid. However, those tumors that acquired radio-iodine in just barely detectable quantities frequently can be induced to acquire a more marked iodine concentration. There are two possible explanations offered to account for this effect of thiouracil in increasing iodine uptake in thyroid carcinoma. The prolonged use of thiouracil may produce a state of iodine depletion so that avidity for iodine of any tissue remotely capable of functioning as thyroid becomes extremely high. A second possible explanation is that propylthiouracil in blocking the conversion of Inorganic Iodine to organic iodine depresses the level of circulating thyroxin causing an increased production of endogenous thyroid stimulating hormone, which has an effect on the neoplastic thyroid tissue. The action of exogenous thyroid Stimulating hormone is more apparent. This merely supplements the patients endogenous hormone in its action on neoplastic tissue.

To produce the desired effect, large doses of propylthiouracil must be given. Thiouracil is usually given in doses of 1.0 to 1.5 grams per day after thyroidectomy for six weeks and every six weeks this treatment is stopped for forty-eight hours

before tracer doses of radio-iodine are given. The problem of how long to continue the use of drugs like propylthiouracil is a very real one but analysis of clinical material frequently solves the problem. A certain number of patients when first seen will have an advanced generalized disease and die shortly after a first tracer study. Likewise, a certain number of patients will have only local disease in the neck and if following thyroid ablation no evidence of iodine concentration can be demonstrated in malignant tissue, then external radiation therapy is indicated, particularly if the lesion is not a very well differentiated carcinoma. Those patients with widespread disease or those with considerable residual disease following radiation therapy should be given propylthiouracil for prolonged periods with tracer doses of radio-iodine at six weeks to three-months intervals, until the patient becomes too ill to continue, or until evidence of iodine uptake is discovered. The prolonged use of thiouracil may produce a severe state of thyroid depression with concomitant symptomatology.

Eventually when iodine concentration is discovered in cancer deposits then therapy with I^{131} should be vigorous. Quantities of 100 to 150 millicuries of radio-iodine are given at two-months intervals, preceded by tracer doses. As long as scanning after tracer quantities of I^{131} indicates that an appreciable percentage is localizing in neoplastic tissue then repeated therapeutic quantities are given.

When such abnormal localization of radio-iodine is discovered, an attempt is made by external counting at different distances to estimate the percentage uptake in each region. The uptake is measured in any residual thyroid tissue that may be left in the neck as is the quantity excreted in the urine. The effective half-life is determined for each area where there is concentration of radio-iodine. An attempt is made to estimate the volume of tumor tissue by clinical and radiological examinations and the dose in roentgens delivered to each abnormal localization determined. Since the clinical estimation of tumor volume is notoriously inaccurate, the figures obtained give only an indication of the order of magnitude of the dose.

What complications may be expected from this sort of treatment? An initial dose of 100 millicuries is commonly given which frequently causes a moderate and transient depression of leucocysts particularly the lymphocytes. Usually with 600 to 800 millicuries given in periods of sixteen to eighteen months there have been no major systematic disturbances, nor has there been any marked depression of bone marrow activity. Local reactions have been observed consisting of local edema and erythema when much tumor tissue was present. Higher quantities of radio-iodine have been given in the same period of time without significant bone marrow depression. Thrombocytopenia and radiation parotitis have been reported following iodine^{131} therapy. Aplastic anemia, and amenorrhea also have been reported. Hyperthyroidism of a transient nature has been mentioned due to destruction of thyroid tissue with release of thyroid hormone. Recently there have been reports of leukemia developing after continued therapy with I^{131} for thyroid cancer.

Reports in the literature state that radioactive iodine therapy is beneficial in approximately 10 to 20 per cent of patients who had total thyroidectomy and who had evidence of metastatic disease. Rather than quote from the literature it may by more beneficial to review the results of radio-iodine therapy in a personally reported series of patients with cancer with metastatic disease. Forty-eight patients were studied with radioactive iodine and in 13 patients there was evidence of enough uptake by tumor tissue to warrant radioactive iodine therapy. Twelve of these patients were treated with radioactive iodine. Table 8 indicates the response to this form of therapy in the 12 patients.

TABLE 8

	Histology	Immediate Response	Result
LOCAL DISEASE IN NECK ONLY: 4 PATIENTS			
Patient 1	Adenocarcinoma with colloid formation	Regression of mass	Alive with residual nodes 2 years, 4 months later
Patient 2	Papillary carcinoma	Regression of mass	Alive and well without disease, 2 years, 6 months later
Patient 3	Hürthle-cell carcinoma	Disappearance of mass	Developed bony metastases 1 years later. Further I^{131} but died 4 years after first treatment
Patient 4	Carcinoma (unspecified)	None	Died within 1 month
DISTANT METASTASES PRESENT: 8 PATIENTS			
Patient 5	Adenocarcinoma with colloid. Later biopsy anaplastic carcinoma	Complete regression of diffuse pulmonary carcinomatosis	Alive and well, no evidence of disease, 3 years
Patient 6	Adenocarcinoma with colloid anaplastic carcinoma at post mortem	Regression of mass. Relief of symptoms	Excellent palliation for 3 years, then died
Patient 7	No histology	Regression of masses	Mass regrew after 6 months. No response to further I^{131}. Give local H.V.T.
Patient 8	Adenocarcinoma with colloid	None	Too early to evaluate
Patient 9	Carcinoma (unspecified)	None	Died within 4 months
Patient 10	Hürthle-cell carcinoma	None	Died within 3 months
Patient 11	None	None	Died within 1 month
Patient 12	Papillary carcinoma	None	Died within 1 month

This series of patients confirms the general experience that relatively few patients with thyroid carcinoma can as yet be successfully treated with I^{131}. Therapy was attempted in 12 patients showing adequate iodine concentration in tumor tissue and only 5 patients had definite evidence of improvement, approximately 10 per cent of the total number of patients seen. There may be, however, an occasional dramatic response to therapy as demonstrated by 1 patient with generalized pulmonary metastases who showed complete regression of demonstrable disease. It is this type of patient with generalized thyroid carcinomatosis who could most benefit by I^{131} therapy, but unfortunately these patients when first seen are frequently in the terminal phase of the disease.

QUESTIONNAIRE

1. When was radioactive iodine first used in the treatment of thyroid carcinoma?
2. Give the physical factors necessary to warrant radio-iodine therapy.
3. Discuss the various ways in which acquisition of I^{131} can be determined and measured.
4. What are the limitations of these methods?
5. Discuss the value of the scintillation counter method.
6. If a nodule shows no iodine uptake why must it be considered as a malignant tumor?
7. What does a low counting rate over a nodule indicate?
8. Discuss the use of external scanning for detection of functioning thyroid carcinoma metastases and for primary thyroid carcinoma.
9. When should total body scanning be done?
10. What type of thyroid tumors can be expected to acquire, concentrate and retain radio-iodine?
11. How would you treat a patient with a wildly growing anaplastic carcinoma of the thyroid?
12. How would you treat a patient with follicular adenocarcinoma?
13. In which cases would you suggest radio-iodine studies?
14. Outline a plan of procedure when thyroid carcinoma is suspected.
15. After thyroid ablation, when should radio-iodine therapy be instituted?
16. Which chemical agents induce or increase uptake?
17. Explain how these agents are able to effect this increased uptake.
18. What is the usual dose of thiouracil to produce the desired uptake effect?
19. How long would you use the drug?
20. What is the therapeutic dose of I^{131} and how often repeated?
21. What complications may be expected from this treatment?
22. Discuss the results of this form of therapy.

Chapter 15

SURGERY OF THE NECK—RADICAL NECK DISSECTION

By A. V. Partipilo

General Considerations.—The term "Radical Neck Dissection" as applied in this chapter refers to the operative procedure intended to eradicate cervical metastatic lymph nodes, the internal jugular vein and contiguous structures that may be involved as a result of carcinoma of the thyroid gland.

The procedure may be a one-stage unilateral neck dissection combined with either homolateral lobectomy or total thyroidectomy; or as a secondary procedure after the primary tumor and thyroid gland have been previously ablated. The procedure is also applicable, with few modifications, in carcinomas originating in the mouth, pharynx, salivary glands, etc.

Indications.—The indications for radical neck dissection in cancer of the thyroid gland are discussed in Chapter 13. Briefly, these may be summarized as follows:

1. The treatment of uncomplicated follicular adenocarcinoma of the thyroid is simple lobectomy. If the thyroid veins appear to be involved, a radical neck dissection is done. In these cases the internal jugular vein should be ligated above the superior thyroid vein and below the inferior thyroid vein and this should be done before starting the dissection of the gland. This segment of the vein is then excised along with the thyroid lobe. It is important to understand that the veins are involved long before there are any evidences of metastatic lymphadenopathy.

2. In papillary adenocarcinoma with metastatic lymphadenopathy, a radical neck dissection is done and combined with total thyroidectomy.

3. The so-called "aberrant thyroid tumor" must be considered as a metastatic invasion of the deep cervical lymph nodes even though a palpable tumor cannot be detected in the homolateral gland. In this case a bilateral thyroidectomy combined with radical neck dissection should be done.

4. In cases with alveolar adenocarcinoma, the anaplastic cancers and in all cancers of the head and neck there should be no clinical or roentgenographic evidence of distant metastasis before a radical neck dissection is contemplated.

5. As a basic principle, radical neck dissection should be done only when there is definite evidence of metastatic cancer in the cervical lymph nodes. The implications of this principle are twofold; first, the mere presence of palpable lymph nodes is not proof of cancerous invasion. These must show clinical and pathological evidence of cancer; second, the acceptance of this principle rules out the so-called "prophylactic neck dissection."

6. Clinical staging is important in determining whether neck dissection should be done (*see* Chapter 13). A radical block dissection should be done on the involved side when definite mobile or slightly fixed lymph nodes are present. Should these be bilateral, still mobile or only slightly fixed, then a bilateral block dissection is indicated. When there is complete fixation of the primary tumor to the adjacent structures or complete fixation of lymph nodes, the patient must then be considered as surgically inoperable.

7. For lesions originating elsewhere in the head and neck, as well as those originating in the thyroid gland, it is important to bear in mind that the decision for or against radical neck dissection must be based upon the findings in each individual case. The broad principles as set forth are merely aids or guides to help in this decision.

Surgical Anatomy.—The extent of dissection necessary for the treatment of cervical lymph node metastases must take into consideration of the fact that carcinoma of the thyroid gland, as well as other cancers of the head and neck, spread by way of the lymphatics, the blood stream, and by direct extension to the

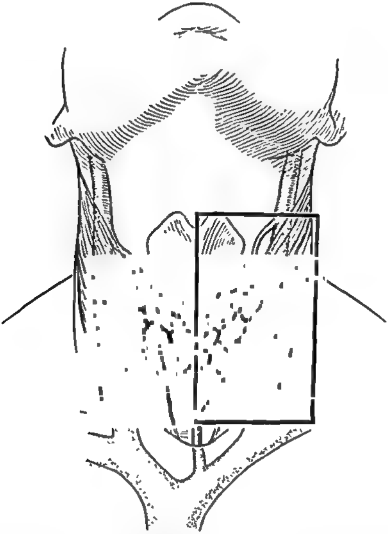

FIG. 96.—Diagrammatic sketch illustrating the extent of radical neck dissection for carcinoma of the thyroid gland.

adjacent structures. From an anatomical viewpoint, the objectives of the procedure are: (1) The removal of the primary tumor, which in this instance requires either a lobectomy or total thyroidectomy; (2) the removal of adjacent structures which may have been invaded. This requires the excision of the sternocleidomastoid muscle, and all of the ribbon muscles; (3) the removal of the lymphatics from the clavicle to the mandible along the internal jugular vein and from the anterior edges of the trapezius muscle to the midline of the neck; (4) because of the possibility of carcinoma of the thyroid to spread by way of the venous channels, the internal

jugular vein and all of its tributaries are removed en masse from the mandible to the clavicle (*see* Fig. 96).

It is rather obvious that in order to accomplish these objectives a thorough knowledge of the anatomy of the neck is essential. A detailed study of this anatomy is found in Chapter 11. The following is a brief review of the cervical lymphatic chain of nodes which will be encountered during radical neck dissection.

The deep cervical lymph nodes are found in close relationship with the internal jugular vein from the base of the skull to the clavicle. They are divided by the omohyoid muscle into two groups: the superior deep cervical nodes and the inferior deep cervical lymph nodes. Each group is further subdivided into a medial and a lateral group.

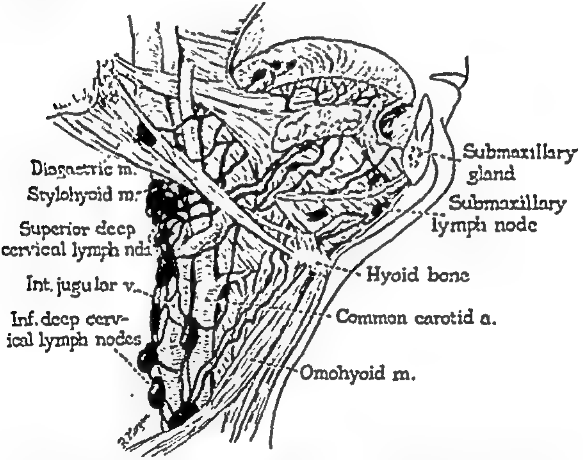

FIG. 97.—Deep lymphatic nodes in the digastric and carotid triangles. Lymph drainage of the tongue is shown. Note the mode of communication between the various nodes. (After Eycleshymer and Jones, Gray's Anatomy, 26th Edition.)

As illustrated in Figure 97, the medial group of the superior nodes lies on the superficial surface of the internal jugular vein in the carotid triangle. The highest of these lie in the area of the union of the common facial vein with the internal jugular vein and are concerned with drainage from the tongue, lips, gums, cheeks and outer part of the nose. Another group is closely associated with the accessory nerve so that this nerve is frequently sacrificed in radical neck dissections. Paresis of the trapezius muscle of the involved side is likely to result. The lowest of the superior group of nodes are located on the internal jugular vein just above the omohyoid muscle.

The superior group of lymph nodes receives efferents from all parts of the head and neck through the following subchains:

free and carefully retracted laterally while the pad of fat and lymph nodes in the posterior triangle are dissected (Fig. 102).

While it is desirable to preserve the spinal accessory nerve, nevertheless, the primary consideration should be directed toward complete removal of all lymph nodes even to the extent of sacrificing the nerve. Following transection of the spinal accessory nerve, a moderate degree of "shoulder drop" occurs due to paresis of the trapezius muscle. The prognosis depends upon the extent to which the cervical

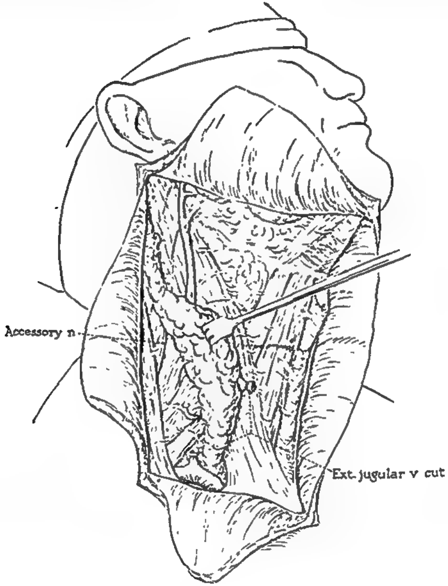

Fig. 102.—Dissection of the posterior triangle. Note the position of the accessory nerve. The external jugular vein has been ligated and cut. The pad of fat and lymph glands are dissected medially.

nerves take over the innervation of the muscle. Early exercises will greatly aid in restoration of shoulder function.

Before proceeding with the dissection, the external jugular vein is isolated at the posterior border of the sternocleidomastoid muscle an inch or so above the clavicle where it is doubly ligated and cut.

A fascial incision is now made in the lower third of the sternocleidomastoid muscle in order to permit the introduction of a finger beneath it and thus isolate the muscle at its sternal and clavicular attachments where it is severed. The muscle

After identifying the phrenic nerve, the posterior belly of the omohyoid muscle is freed and cut with short sweeps of the scalpel. Again, each bleeding point is immediately ligated. The sternohyoid and sternothyroid muscles are likewise severed from their attachments. The severed ends of these muscles, together with the ends of the sternocleidomastoid and omohyoid muscles, are now encircled with a strong silk suture, thus converting them into a single bundle which can easily be manipulated during the remaining portion of the neck dissection (Fig. 103).

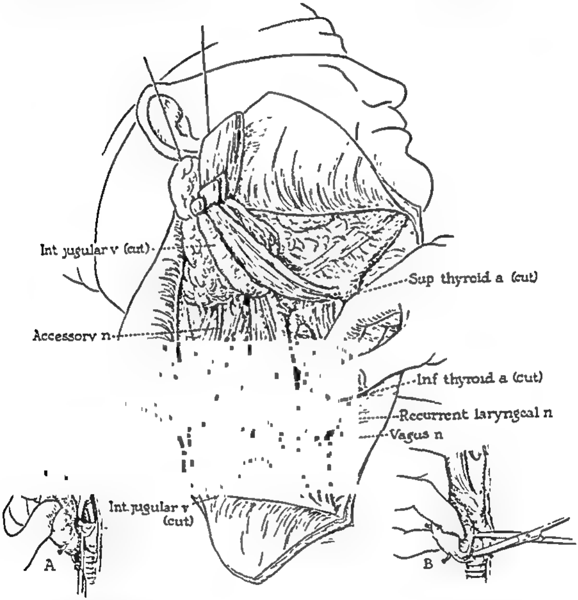

FIG. 104.—The internal jugular vein has been cut and included in the encircled bundle of muscles. The blood supply of the thyroid gland is being ligated and the gland is removed.

As illustrated in Figure 104, the encircled stump with the pad of fat and lymph nodes is reflected upward exposing the carotid sheath, the phrenic nerve, the cervical trunks of the brachial plexus, the muscles forming the floor of the posterior triangle, the thyroid gland and tracheal rings.

The next important step is the ligation of the plainly visible internal jugular vein, located within the carotid sheath. The vein must be isolated and tied without injuring the vagus nerve, the common carotid artery or the thoracic duct. In this area of the neck, the right internal jugular vein is somewhat separated from the

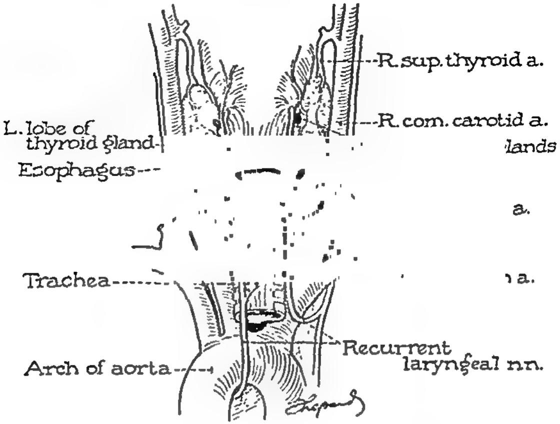

FIG. 105.—The relationship of the recurrent laryngeal nerve to the thyroid gland.

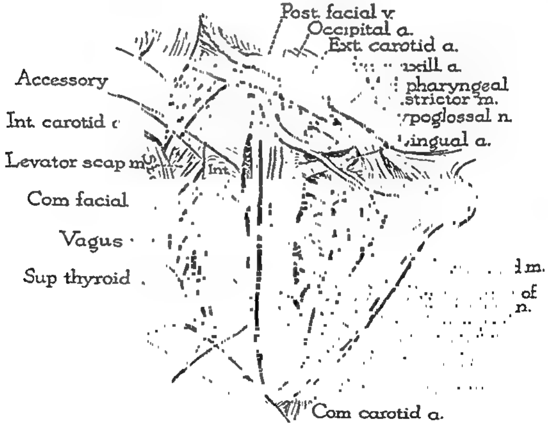

FIG. 106.—Deeper dissection of the carotid triangle illustrating the various structures which must be avoided.

After reflecting the digastric muscle upward, the internal jugular vein is doubly ligated with a strong silk suture and cut. The entire mass of tissue is then discarded, thus completing the neck dissection. The posterior belly of the digastric and the stylohyoid muscles may be sacrificed if these appear to be attached to the metastatic nodes, otherwise retraction upward is sufficient to permit adequate exposure for isolation of the internal jugular vein.

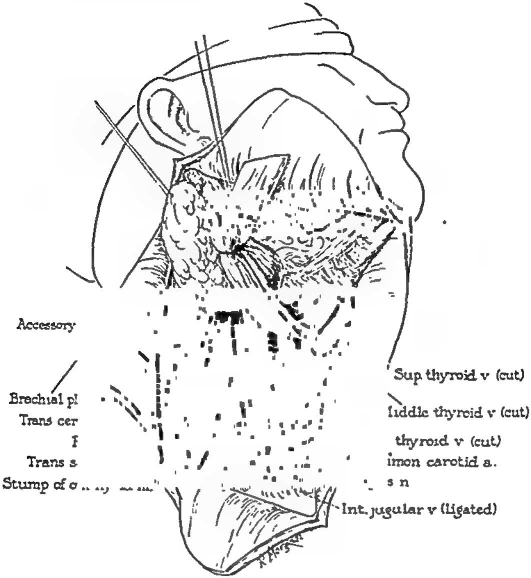

Fig. 107.—The completed radical neck dissection.

Closure of the Wound.—Before closure all bleeding must be perfectly controlled. Since a large surface has been exposed, one should expect a considerable amount of serous drainage. A Penrose drain should be sutured with plain four 0 catgut to the upper end of the neck, to the stump of the sternocleidomastoid or to the digastric muscle, and be allowed to emerge from the angle of the collar incision. The skin wound is now closed with either silk or with metal clips. Pressure dressings are applied with care being taken not to apply undue pressure on the trachea. Judicious use of pressure will reduce the amount of drainage; nevertheless, there will be a copious amount of drainage for the first few postoperative days. For this reason the dressings should be changed daily. The drainage tube should be left in place

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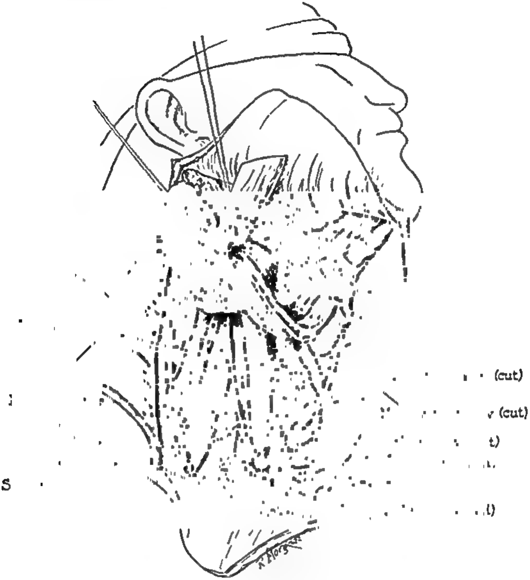

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until the fourth or fifth day when a tract has been well established to insure adequate drainage even though the tube is removed.

Postoperative Treatment.—Patients who have undergone radical neck dissection present specific postoperative problems which demand special nursing care in addition to that given to any patient who has undergone a major operation. The most urgent and immediate postoperative problem is the constant nursing care to see that the patient's breathing is unobstructed as long as he remains in the unconscious state. The endotracheal tube should not be removed before the patient's reflexes have returned and the patient throws out the tube voluntarily. Prior to extubation the nurse should suction out any secretions which may have accumulated.

QUESTIONNAIRE

1. Give a broad definition of the term "radical neck dissection."
2. When is this procedure indicated in follicular adenoma of the thyroid gland?
3. When is it indicated in the presence of papillary adenocarcinoma?
4. How would you consider and treat "aberrant thyroid tumor"?
5. When would you do this operation in cases of alveolar and anaplastic cancers?
6. Would you do a "prophylactic neck dissection"? Explain.
7. Discuss the importance of clinical staging as an aid in determining the whether this operation should be done.
7. What are the objectives of the operation?
8. Describe the lymph nodes of the neck.
9. Describe the technique of radical neck dissection.
10. What is the most urgent postoperative problem?

Chapter 16

SURGERY OF THE NECK—TUMORS OF THE CAROTID BODY

By A. V. Partipilo

General Considerations.—The carotid body was first described by Von Haller in 1743 as the "ganglion minimum." Andersch, in 1797, gave an excellent description and named the body at the bifurcation of the common carotid artery as the "ganglion intercaroticum." No specific interest was attached to this organ until 1862 when Luschka published a splendid article describing in detail its microscopic anatomy. Since then numerous and extensive studies have been made, and though many characteristics have been cleared, there are many points which are as yet obscure.

Reigner, in 1880, is accredited with being the first to have recognized and removed a carotid body tumor, first described by Marchand in 1891. Tumors of the carotid body are noteworthy for their extreme rarity. Very few surgeons have had to operate or report more than one case, nor more than 47 cases from a clinical group. Even the busiest surgeon or pathologist never becomes entirely familiar with these tumors and must depend upon the collected experience of others. It is therefore rather difficult for any one surgeon to pass judgment or to become dogmatic on the subject. For this reason a review of the various phases of this subject is covered in this chapter.

Anatomy and Embryology.—The carotid body is a paired organ situated posterior to the common carotid artery at its bifurcation. It is variable in size, ranging from 5 to 7 millimeters long and 2½ millimeters thick. Its location is not absolutely constant, at times being in the middle of the common carotid and on other occasions it lies on the medial posterior surface just at the bifurcation of the artery. The gland has a fibrous capsule and is connected to the wall of the carotid artery, usually the external, by a fibro-vascular pedicle called the ligament of Meyer. It is through this pedicle that it receives its nutrient vessels. Depending upon its blood supply, the normal gland varies in color from greyish-red to reddish-brown. The nerve supply to the gland is from branches of the superior cervical sympathetic ganglion, the superior laryngeal, the glossopharyngeal, and sometimes from the vagus nerve.

Histologically, the chief cells observed are pale staining epithelial-like cells with pale nuclei which are eccentirc, hyperchromatic, and frequently vacuolated. The cells are large, and polygonal and the cytoplasm is abundant and finely granular. Physiologically, the glands do not have any known function and are not considered to have any internal secretion.

There is considerable difference of opinion as to the embryological origin of the carotid body gland Three theories which have been strongly advocated may be grouped under the following headings: (1) Epithelial; the advocates of this theory believe that the gland is derived from the pharyngeal epithelium. There is no evidence to support this theory. (2) Vascular theory; the proponents of this theory believe that the body is derived from either the perithelium or endothelium of the

blood vessel wall. Boyd showed that the carotid body originates in the adventitia of the artery and that tumors arising from this gland show this same relationship to the vessel. Smith and Gault classify carotid body tumors as a variety of endothelioma, more often spoken of as perithelioma because of the striking perithelial arrangement of their cells. Morfit, Swan, and Taylor, in 1953, in an excellent and extensive review of the subject, concluded that the confinement of these tumors to the adventitial layers of the artery even in the advanced stage permits the removal of the tumor without damaging the artery. (3) Finally there are advocates who believe that the gland is derived from the sympathetic ganglion cells of the intercarotid plexus.

Pathology.—Grossly, the tumor appears to be reddish brown, lobulated with well-encapsulated nodules. In a series of 47 cases seen at the Mayo Clinic and reported by Petter *et al.*, the size of the tumors averaged 4.5 by 3 centimeters and the largest measured 8 by 7 by 5 centimeters. Microscopic examination of the tumors revealed two predominant patterns; one, an organoid type and the other a peritheliomatous type. The organoid type was present in all tumors whereas the peritheliomatous pattern was noted in 16 specimens. The organoid type showed a striking resemblance to the normal structure of the carotid body. The tumor cells were large, polyhedral, epithelioid cells of fairly uniform size. The cytoplasm was abundant and faintly granular. The nucleus was oval, eccentric and had a well defined nuclear membrane. The nuclear chromatin appeared in small clumps and frequently more than one nucleolus was present. In the peritheliomatous pattern, the islands of cells were smaller and less distinctly demarcated than in the organoid type. The cells were identical with those in the organoid type, except that in a few instances they assumed a roughly crescentic shape. Hyalinization of connective tissue is a prominent feature of the peritheliomas.

The consensus of opinion is that the great majority of these tumors are benign and that malignant manifestations occur in about 15 per cent of cases. They are usually slow growing, well encapsulated and all evidence points to rare metastases. Very definite malignancy, as shown by active mitosis, cellular variation with giant cells, invasion of the capsule and the like, was present in 10 of the 20 cases reported by Harrington *et al.* They suggest that these tumors be regarded as neoplasms of low grade malignancy with potential powers of invasion and metastases. According to Lahey and Warren, local metastases to the regional nodes have been observed but distant metastases, suspected in several instances, including one of their own cases, have not been verified. Morfit, *et al.*, reported a case in which distant metastases in the lung ultimately caused death eighteen months after the original tumor had been removed.

Pettet *et al.*, are emphatic in their opinion that these tumors are not malignant and in none of their cases did they find evidence of metastases.

Diagnosis.—The diagnosis of a carotid body tumor is very difficult and seldom made. Positive diagnosis cannot be made except by a microscopic examination of the specimen. This is because there are 4 laterally located tumors situated in the neck which have common characteristics. These tumors are: the carotid body tumor, branchial cyst, solitary "aberrant" thyroid gland and neurofibromata. In all cases there is a visible tumor situated in the anterior cervical triangle of the neck. The growth is single, discrete, movable, and usually painless. A carotid body tumor may vary in size from that of a hazel-nut to that of a goose egg. It is slow growing and can be seen to be rather deeply situated underneath the sternocleidomastoid

muscle. The tumor is ovoid or round, firm but elastic, and because of its great vascularity it can be compressed and reduced in size temporarily. The tumor is never attached to the skin and the latter is freely movable over the mass. As the tumor enlarges it does so in an upward direction toward the angle of the jaw and may even bulge into the pharynx thus interferring with swallowing. One outstanding feature of a carotid body tumor, which has been overemphasized, is its mobility in a lateral direction and immobility in the vertical direction because of its attachment to the carotid artery. Occasionally, a bruit or thrill can be elicited and a transmitted pulsation from the carotid artery can be seen.

Branchial cysts are superficially situated and have a tendency to enlarge outward and become more and more superficial. Their greatest diameter is generally below the level of the carotid bifurcation, and the lower they are located the more superficial they become. A distinguishing feature of a branchial cyst is that when traction is made upon it, or on its fistulous opening, the sinus tract can be felt underneath the skin extending from the angle of the jaw and disappearing under the digastric muscle where the tract proceeds deeply to become connected with the lateral pharyngeal wall in the region of the tonsil.

Tumors of the lateral "aberrant" thyroid gland are generally multiple and may be found in any or all triangles of the neck. In the occasional instance when it originates as a single discrete tumor in the anterior triangle, it may be rather difficult to differentiate from a carotid body tumor. When they are multiple, there is no question of them being confused with carotid body tumors, but they are then more often mistaken for enlarged lymph nodes. A distinguishing feature of a lateral "aberrant" thyroid tumor is that it is seldom found under the angle of the jaw.

Neurofibromatas can occur anywhere and occasionally may be found in the carotid triangle and thus become confused with carotid body tumors. Because of their origin from deeply situated nervous tissue, it may be impossible to distinguish neurofibromatas from other tumors of the neck.

Morfit *et al.*, believe that on the basis of chance alone, the most common explanation of an asymmetrical enlargement in an adult is a metastatic growth from an intraoral primary cancer. Therefore, they stress the importance of starting the examination by thorough exploration of the mouth and pharyngeal cavities. If these are unproductive, attention is then directed to the mass itself. In their experience, physical findings when used as a basis for specific diagnosis are attended with such a high degree of inaccuracy as to hardly justify making a diagnosis except by histologic proof (biopsy). In their hands, aspiration biopsy has been found to be extremely valuable resulting in a correct diagnosis in 90 per cent of instances in which it was employed. In the final analysis surgical exploration is recommended as this is the only method which can establish the true nature of the tumor.

Other symptoms that may occur from a carotid body tumor are those due to pressure effects on the vagus, cervical sympathetic, and recurrent laryngeal nerves. Thus, a patient may complain of any of the following symptoms: hoarseness, dyspnea, tinnitus aurium, headache, dilatation of the pupils, fainting spells due to pressure on the vagus and even Stokes-Adams syndrome.

Treatment.—The problem of what to do with carotid body tumors is not an easy one to resolve. With few exceptions, surgeons have a very limited knowledge of the subject especially the personal experience necessary to evaluate the best course to follow for each individual case. We are dealing with a slow growing, painless, asymptomatic tumor which is benign in 80 to 85 per cent of cases. The

tumor is firmly attached to the common carotid artery and experience has shown that ligation of this artery with removal of the tumor is attended with a mortality rate of more than 30 per cent. Even if the patient survives, cerebral complications are apt to occur. In 1929, Bevan came to the conclusion that a carotid body tumor should not be removed when it is necessary to ligate the carotid arteries since a malignant growth involving the arteries offers very little prospect of a permanent cure even by the most extensive operation. However, if the common carotid and the internal carotid can be saved, he felt justified in removing a benign tumor.

In 1947, Lahey and Warren believed that when the pre-operative diagnosis of a carotid body tumor has been correctly made, or when a tumor is demonstrated in exploration to surround the internal, external and common carotid arteries completely, removal by ligation of the arteries should be done only when biopsy demonstrates the tumor to be truly malignant. They further stated that even then it should be removed only when the patient has been able to work up to complete carotid compression for a period of ten minutes three times a day without symptoms, and when friends and family are well aware of the risks involved.

In 1953, Pettet *et al.*, came to the conclusion that because of the high mortality and morbidity rates associated with ligation of the carotid arteries and the slow-growing benign nature of these tumors, no attempt should be made to remove the tumor if it necessitates ligation of the common or internal carotid artery. They do not agree with the generally accepted opinion that these tumors are malignant even in some instances.

Morfit *et al.*, do not believe that carotid artery ligation can be made safe by preliminary intermittent digital compression on the artery, or by stellate ganglion block, nor that gradual preligation of the vessel will be effective. In their opinion, no method is currently available to make carotid artery ligation safe and that in view of the high incidence of fatal, crippling or unpleasant sequelæ when this vessel is ligated in the treatment of carotid body tumor, the procedure is unjustified without restoration of arterial continuity.

Conley, in 1953, suggested and performed an end-to-end anastomosis of a free autogenous graft to the internal and common carotid arteries when it became necessary to excise these vessels. This procedure requires further study and investigation before acceptance. It should be of value in those cases where the surgeon has no other choice.

Gordon-Taylor, in 1940, cautioned the surgeon against resecting the carotid bifurcation and expressed the view that even when the arteries appear to be almost imbedded in the growth, meticulous care and painstaking dissection may sometimes reveal a "white line" whereby the continuity of the main arteries may remain undisturbed or at least structurally inviolate. Morfit *et al.*, in a consideration of their experience in 12 cases and after a thorough study of the problem, have come to the same conclusion. It is their belief that because of the embryological origin of the carotid body and the confinement of these tumors to the adventitial layers of the artery even in the advanced state, successful removal of the tumor can be achieved without damaging the artery. Furthermore, they state that in the event that the lumen of the artery is inadvertently entered in the course of the removal, ligation should never be resorted to because there is no method by which such a step can be made safe for the patient, regardless of any preoperative or postoperative adjunctive measures. Restoration of continuity of the arterial stream by means of simple closure of the tear, primary end-to-end anastomosis, and insertion of an

arterial autograft or venous autograft as advocated by Conley are recommended in that order as solutions to this complication. We subscribe to these views and observations of Morfit *et al.*, as sound surgical judgment. The operative description described herein follows closely their views.

Technique.—The patient is draped and placed on the operating table in the same position as for a thyroidectomy except that the head is turned to the opposite side. The type of incision is determined by the size of the tumor. If small or of moderate size a circular collar incision is made along a crease of the neck. This incision extends from about the middle of the sternocleidomastoid muscle to a point on the medial border of the component muscle on the other side. The height of the incision is at a point where the common carotid normally bifurcates, *i.e.*, at the level of the cricoid cartilage. The skin, superficial fascia and platysma are incised

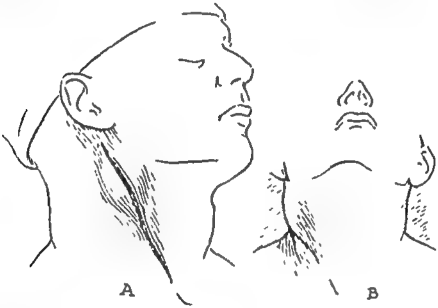

FIG. 108.—*A*, Longitudinal incision along the anterior border of the sternocleidomastoid muscle from the angle of the jaw to the sternal notch. *B*, Appearance of the tumor in the anterior position.

and both flaps are then dissected upward and downward respectively as in thyroidectomy. This exposes the deep cervical fascia and the underlying sternocleidomastoid muscle. The deep fascia along the anterior border of the sternocleidomastoid muscle is then divided and the omohyoid muscle is encountered at the lower border of this facial incision. This muscle is retracted downward, or it may be divided if additional exposure is required. The sternocleiodmastoid muscle is retracted outward. The posterior belly of the digastric muscle is seen at the upper portion of the incision and this muscle is retracted upward. With these maneuvers the carotid tumor and the various branches of the carotid artery are fully exposed.

The circular collar incision is done only when the size of the tumor is small, however in most instances it does not offer adequate exposure and for this reason a longitudinal incision is preferred.

A longitudinal incision is made along the anterior border of the sternocleidomastoid muscle. For a very large tumor the incision may be made from the angle of the jaw to the sterno-clavicular angle (Fig. 108). This incision allows a much better exposure of the omohyoid and posterior belly of the digastric muscles. If the tumor is extremely large it may be necessary to completely incise across the

omohyoid muscle in order to obtain good exposure of the common carotid well below the tumor.

After the incision has been made, the skin flaps are reflected and an incision is made in the deep fascia along the anterior border of the sternocleidomastoid muscle. The omohyoid muscle is either retracted downward or cut across, the sternomastoid is retracted outward and the posterior belly of the digastric is retracted upward.

As illustrated in Figure 109 the dissection thus far has uncovered the carotid triangle with its contents. The common carotid artery, the internal jugular vein and the vagus nerve are enclosed within its sheath. The common carotid artery is now localized as it crosses the carotid tubercle, the costal process of the sixth

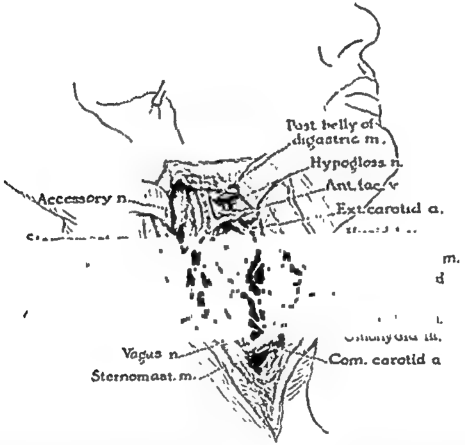

FIG. 109.—The sternocleidomastoid muscle is retracted outward, the omohyoid downward or cut across and the posterior digastric muscle upward. The illustration shows the content of the carotid triangle and the carotid body tumor.

cervical vertebra. At this time it is important that the surgeon bear in mind the anatomical relationship of the various structures in the carotid sheath and their relationship to the various structures in the immediate vicinity.

There are three structures in the carotid sheath; the internal jugular vein, located laterally; the common carotid artery found medially; and the vagus nerve situated posteriorly in between the artery and the vein. Along the front of the artery and outside of the sheath is the descending branch of the hypoglossal nerve.

The carotid sheath is now carefully incised avoiding the descending hypoglossal nerve and the common carotid artery is freed in its entire circumference well below the tumor. After this has been done a safety tape is applied as illustrated in Figure 110. If possible, the internal and external carotids are similarly treated. The purpose of the safety tapes is to temporarily control excessive bleeding in the event

Fig. 110.—The common carotid artery is freed from the carotid sheath well below the tumor. Saftey tapes have been applied on the common carotid and the internal and external carotid arteries.

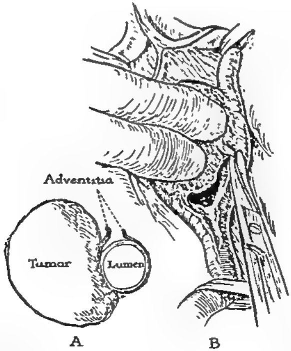

Fig. 111.—The dissection of the tumor is started at a point away from the tumor in a plane between the adventitia and the media of the common carotid artery. Insert (*A*) illustrates the position of the tumor and the adventitia of the artery.

it occurs during the process of tumor dissection. The carotid body tumor is traversed throughout its entire substance with considerable vascular channels and when these are cut across they will bleed rather briskly, and may suggest that a rent has been made in the main artery. This complication can be treated with a figure-of-eight transfixion suture while traction is made on the tapes to control the bleeding. If the main artery is actually cut, the surgeon should endeavor to sew it with fine silk threaded on a Cambric needle or, this failing, one may try an actual primary anastomosis between the internal and common carotids. In the event that neither of these is possible, Morfit suggests the use of a stored homograft or a freshly obtained venous graft.

The dissection of the tumor is started at a point away from the tumor in a plane between the adventitia and the media of the common carotid artery (Fig. 111). With meticulous and painstaking care the tumor can be removed without injury to the arteries. The plane of the dissection can be delineated by injecting 1 per cent procaine underneath the adventitia. This injection also has the advantage of nullifying any stimulus to the carotid sinus with the attendant bradycardia that sometimes occurs. For this condition the stellate ganglion can also be injected at this time.

After the tumor has been completely removed all bleeding points are controlled and the wound is closed with a small Penrose drain being inserted in the lower angle of the wound. The postoperative care of the patient is the same as for any major operative procedure on the neck.

QUESTIONNAIRE

1. Who was the first to describe the carotid body?
2. Who was the first to recognize a carotid body tumor?
3. Where is the carotid body located?
4. Describe the histological characteristics of the carotid body.
5. What is the ligament of Meyer?
6. Discuss the embryological theories as to the origin of the gland.
7. Describe the gross appearance of a carotid body tumor.
8. Describe the microscopic appearance of the tumor.
9. Discuss the incidence of malignancy of these tumors.
10. What are the diagnostic features of this condition?
11. What are the most common conditions in the neck from which it must be differentiated?
12. Discuss the treatment of carotid body tumors.
13. What is the mortality rate following ligation of the carotid artery?
14. Discuss the surgical technique for the removal of the tumor.
15. Describe the surgical anatomy involved in this procedure.
16. What would you do if the carotid artery is accidentally severed?

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Chapter 17

SURGERY OF THE NECK—THYROGLOSSAL DUCTS AND CYSTS

By A. V. Partipilo

General Considerations.—The thyroglossal duct is a tubular embryonal outgrowth originating at the foramen cecum of the tongue, descending downward behind the body of the hyoid bone, then passing in front of the larynx and trachea to reach the isthmus of the thyroid gland. During fetal life it is canalized but later it becomes obliterated and disappears. When the duct fails to become completely obliterated, or remnants of tissue between the origin of the duct and the isthmus persist, any number of conditions may develop.

The most common developmental defect is a thyroglossal cyst usually seen in the anterior midline of the neck. Rarely, if the upper end of the duct remains unobliterated, a cyst may form at the base of the tongue or just immediately below it. If the cyst should break into the skin a sinus with an external opening may develop and persist as a fistula. The fistulous opening on the skin is generally found in the midline; however it may be found at some distance laterally if an underlying infection or abscess has pointed from the midline. This may make it difficult to differentiate the condition from a branchial fistula. Aberrant thyroid tissue may also be found in the midline of the neck associated with a persistent thyroglossal duct and, unless the duct is removed with the aberrant tissue, complete recovery is not possible. Finally, the lower end of the duct may also persist as the pyramidal lobe of the thyroid gland.

If there is faulty descent of the thyroid enlage and duct, the gland may be found ectopically situated anywhere between its normal location and the base of the tongue.

The diagnosis of thyroglossal anomalies is based upon the finding of a cystic mass or sinus in the midline of the neck. The characteristic feature of a thyroglossal cyst is that it moves with deglutition. For this reason it may be mistaken for the thyroid gland if the cyst is within the region of the isthmus of the thyroid or an ectopic thyroid may be confused with a cyst.

The cysts vary in size from that of a marble to that of a hen's egg. They are smooth, well defined and the smaller ones move rather easily between the examining fingers. By careful manipulation, the tract of the duct can be felt leading to the hyoid bone. As previously mentioned, they move with deglutition, however; they are painless unless infection is superimposed. Mention should be made of the rare finding of a cyst with a persistent thyroglossal duct which opens into the foramen *cecum* of the tongue. By applying pressure over the cyst, fluid can be expressed into the pharynx. A fistula of the thyroglossal cyst is generally due to secondary rupture of an infected cyst or as a result of surgical incision and drainage of an abscessed cyst. In these infected cases the sinus opening may be found at quite a distance and laterally placed from the midline of the neck. An inflamed cyst is usually painful and motion is limited which may make the diagnosis rather difficult.

Thyroglossal anomalies must be differentiated from the following conditions in the neck: (1) Enlarged inflammatory lymph nodes as a result of infected teeth, lips or chin. These are more anteriorly placed than a cyst and the primary source of their causation is generally evident. (2) Lipomata found in the midline or just lateral to the midline may cause some confusion; however their soft lobulated consistency rules out cysts. (3) An aberrant placed thyroid may be difficult to differentiate from a persistent thyroglossal duct and cyst. Since the treatment of this condition and of cyst is the same it does not present any problems. (4) Ectopic thyroid may be mistaken for a cyst. In this contingency, it is important to make certain that a normally located thyroid gland is present before attempting to make any surgical excision. (5) Discrete tuberculous glands are usually located laterally and much deeper. (6) Dermoids and sebaceous cysts are found within and fixed to the skin.

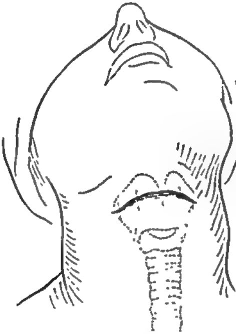
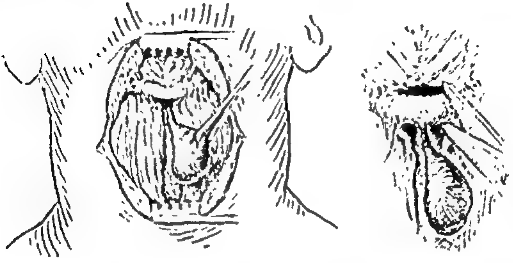


FIG. 112.—The skin incision.

Treatment.—The surgical principle involved in the treatment of thyroglossal cyst or sinus is the complete removal of the tract to the foramen cecum along with the removal of a portion of the hyoid bone. The method described by Sistrunk is favored by this author and is described herein.

The position of the patient on the operating table is the same as for thyroidectomy. Endotracheal anesthesia is the agent of choice. A transverse incision (Fig. 112) about [illegible] inches long is made along a normal crease of the skin over the prominent portion of the presenting cyst. The incision is then developed through the subcutaneous fat and platysma muscle. The skin flaps are then dissected upward and downward exposing the deep cervical fascia. The cyst is seen underneath the deep fascia joining the sternohyhoid muscles. A longitudinal incision is now made in the deep cervical fascia to expose the cyst. By careful dissection, the fascia and the sternohyoid muscles are freed from the cyst. With slight traction on the cyst the fistulous tract is then seen to course upward toward the hyoid bone. To obtain better exposure it may be necessary at this time to divide any tissue covering the mylohyoid muscles. With sharp dissection, about 1 to $1\frac{1}{2}$ centimeters of the

central upper and lower borders of the hyoid bone is freed from its muscular attachments (Fig. 113*B*). The bone is now grasped with an Allis forceps and pulled to one side of the midline and forward. As illustrated in Figure 114 the hyoid bone is divided at each end so that about one centimeter is removed.



A B

FIG. 113.—*A*, The cyst and tract dissected up to the hyoid bone. *B*, The muscular attachments to the hyoid bone have been severed.

FIG. 114.—The hyoid bone has been cut and the cyst with the tract is pulled forward.

In order to remove the tract completely an assistant, or preferably the anesthetist, pulls the tongue forward and with the index finger placed on the foramen cecum, he pushes it forward and upward thus shortening the distance between the foramen and the hyoid bone (Fig. 115). Now, without any attempt to isolate the duct, the tract and the tissue surrounding it for a distance of about 3 millimeters on all sides, are cored out through the muscles of the tongue to the mucous mem-

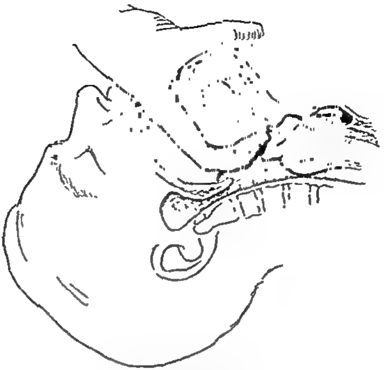


FIG 115.—Illustrates the position of the assistant's finger on the foramen cecum and its relationship to the duct.

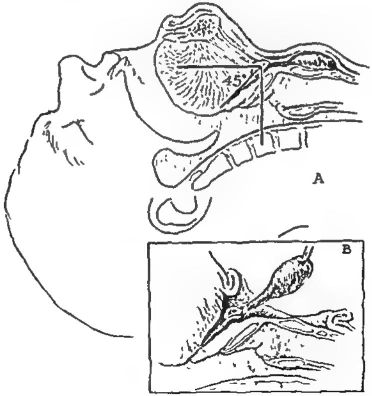


FIG 116.—Illustrates the angle of the tract; the insert shows the dissection of the tract down to the ... ane.

brane of the foramen cecum where they are cut off without opening the mucous membrane of the mouth (Fig. 116*B*). In carrying out this latter dissection it is important to bear in mind the direction of the thyroglossal duct. With the patient in the suggested position, the line of the duct corresponds to a line drawn at an angle 45 degrees backward and downward toward the foramen cecum through the inter-

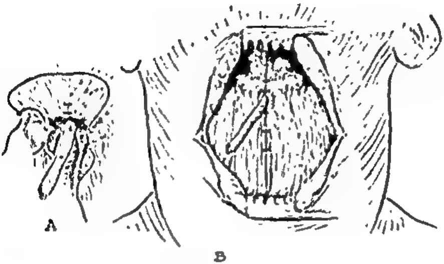


FIG. 117.—Insertion of drainage tube and the suturing of the hyoid bone.

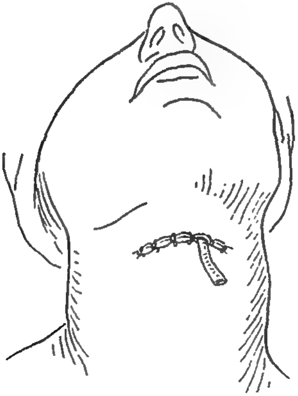


FIG. 118.—Closure of the skin wound and drainage tube.

section of a line drawn horizontally and perpendicularly to the center of the hyoid bone as illustrated in Figure 116*A*.

The opening through the muscles of the tongue and the deep cervical fascia is closed with interrupted chromic 0 catgut. Sistrunk advocates suturing the two ends of the hyoid bone; however, this is not always necessary since they seem to come together without tension. A small Penrose drain is now inserted down to

the mylohyoid muscle and the subcutaneous tissue. The platysma and skin are approximated in the same manner as in the closure of a thyroid incision. The drain can be removed in twenty-four to forty-eight hours.

QUESTIONNAIRE

1. What is the embryonal origin and extent of the thyroglossal duct?
2. After birth what happens to the duct?
3. If the duct fails to obliterate, enumerate the various conditions that may develop.
4. Give the symptoms and physical findings of a thyroglossal cyst.
5. What is its characteristic feature?
6. What is the cause of a thyroglossal fistula or sinus?
7. Discuss the differential diagnosis.
8. What is the surgical principle in the treatment of thyroglossal cysts and fistulæ?
9. Describe the technique for excision of a thyroglossal cyst.

Chapter 18

SURGERY OF THE NECK—BRANCHIAL CYSTS

By A. V. Partipilo

General Considerations.—Branchial cysts or clefts, also known as congenital cysts, are probably better designated as lateral neck cysts or fistulæ. A branchial cyst results from incomplete obliteration of a branchial cleft, the unobliterated portion becoming distended with secretions from its epithelial lining. A branchial cyst is usually superficially located in the anterior triangle and is situated anterior to the sternocleidomastoid muscle. It is found higher placed than the cutaneous opening of a fistula. Although the cyst may be found anywhere along the anterior border of the sternocleidomastoid muscle, it is usually located at the middle third of the muscle.

Branchial fistulæ may be classified into three types: (1) The complete fistula having both an internal and external opening. The internal opening may be found near the base of the tonsil or along the posterior tonsillar pillar. (2) The second type is the incomplete external fistula, in which the proximal portion is obliterated and the distal portion opens into the skin. This is the common variety. (3) The third type is the incomplete internal fistula, where the internal opening is in the pharynx and the distal portion of the sinus is obliterated. This is the rare type.

Branchial cysts usually appear between the ages of ten and twenty and the condition occurs predominantly in the female. They grow slowly and painlessly. The patient is unaware of the condition until the "lump" in the neck becomes visible or a draining sinus draws the patient's attention. The tumors are generally soft and fluctuant and are located anterior to the sternocleidomastoid muscle. They may be found anywhere from the angle of the jaw to just above the clavicle but are usually found at the middle third of the sternomastoid muscle. They may occur on either side of the neck and on occasion the tumor may be bilateral. The cysts vary considerably in size from that of a marble to that of an orange. Their consistency varies with the amount of pressure and fluid in the cyst. They are usually soft and fluctuant and are limited in motion because of their attachment to the overlying tissue. This is particularly true if an infection has occurred. If the size of the tumor varies from time to time, the probabilities are that this is an internal incomplete cyst with an internal opening in the pharynx.

The most prominent symptom of a branchial fistula is a draining sinus on the skin. This opening is usually not greater than few millimeters; very often it will not admit even the smallest probe. A mucoid discharge is pathognomonic of the condition. A microscopic examination of this discharge will reveal squamous epithelial cells from the desquamated lining of the sinus. Secondary infection is a frequent complication and when it has occurred the discharge is purulent in character and the opening shows signs of inflammation.

The diagnosis of branchial cyst and fistula is frequently difficult and must be differentiated from the following: (1) Carotid body tumors: these are deeply

situated underneath the sternocleidomastoid muscle. They are firm, elastic tumors and the skin over the tumor is freely movable. (2) Dermoid cysts or cartilaginous rests: these are usually differentiated by the finding of hair, cartilage, or other foreign substance in the tumor. They do not, as a rule, have an external opening. (3) Cystic hygroma: these are usually transparent and can be transilluminated. They are found in the posterior triangle. (4) Thyroglossal cysts and ducts: these invariably occur in the midline, are attached to the hyoid bone, and shift position with deglutition. (5) A draining sinus from a tuberculous lymphadenitis may be confused with a branchial fistula. The character of the discharge and the finding of matted lymph glands in the neck, and tuberculosis elsewhere is sufficient to suggest the diagnosis. (6) Lipomas should not offer any difficulty. They are as a rule lobulated, firm and aspiration reveals no fluid content. (7) Deep cervical abscesses *are characterized by pain, tenderness and other symptoms of a local and general* inflammatory reaction. It may be difficult to differentiate this condition from an infected branchial cyst without an external opening, except that in the latter condition the inflamed tumor is more superficially situated. (8) Finally, branchial cysts may be confused with various metastatic neoplasms, Hodgkin's disease, lymphatic leukemia and lymphosarcoma. These can be differentiated by blood studies or by biopsy of the tumor.

Anatomy and Embryology.—In the second or third week of intra-uterine life a series of branchial or pharyngeal pouches are formed from which the future structures in the neck are developed. With the formation of these pouches internally a series of clefts appears externally. These correspond to the first four pouches and are called pharyngeal or branchial clefts.

From the first pharyngeal is developed the auricle of the external ear and the cavity of the cleft becomes the external acoustic meatus. The first pouch forms the lateral portion of the upper lip and maxilla, the lower lip and mandible, and the body of the tongue. The second pouch develops into the stylohyoid ligament and the lesser cornu of the hyoid bone. The body and the great horn of the hyoid bone are derived from the third pouch. The remaining pouches make up the rest of the soft parts of the neck. The first cleft forms the external auditory canal and lobe of the ear; the second cleft, the tonsillar fossa; the third cleft, the thymus gland; and the fourth cleft, the thyroid gland. The branchial clefts unite across the median line and become obliterated by the end of the seventh or eighth week of gestation.

A branchial cyst is the result of incomplete closure of a branchial cleft, the unobliterated portion becoming filled with secretion. Embryologically, it is possible for a branchial cyst to result from failure of any of the four clefts to become obliterated. Defects in the first branchial cleft are extremely rare and no case was reported in the literature up to 1923. In 1953, Rankow and Hanford reported 3 cases of first branchial cysts and fistulæ out of 167 cases of all types which were seen during a twenty-year period at the Presbyterian Hospital, New York. According to these authors, the tracts were all situated anterior and superior to the posterior belly of the digastric muscle and the carotid artery. Each of these was satisfactorily treated at operation by a single tran incision from the midline to the tip of the mastoid process. An ellipse included the external fistulous opening. The tract was excised just sh rnal communication with the external auditory can. 'oid injury anic memb a cartilaginous defect in t canal was Rankow, found

that in the presence of a tentative diagnosis of a branchial defect in the neck, methylene blue or lipiodol visualization of the fistulous tract in association with direct examination of the external auditory canal are useful aids to arrive at a correct diagnosis of congenital anomalies of the first branchial cysts.

The most extensive study of the embryology and explanation of the etiology of branchial cysts or fistulæ has been made by Wenglowski and Meyer. Wenglowski's theory is based upon the study of 246 cadavers and 75 embryos. In substance, this theory proposes that branchial defects originate from the thymic duct which is derived from the third pouch. Others, including Shedden and Ladd disagree with this theory. Ladd and Gross rule out the fourth and fifth clefts as precursors of fistulæ because such tracts would necessarily follow a course below the corresponding fourth branchial artery, or its derivative, namely, the aortic arch on the left or the subclavian artery on the right side. Since no branchial fistula has been reported in these positions, the fourth and fifth clefts can be ruled out as being responsible for these clefts. If the fistula was derived from the third cleft, it would have to follow a course posteriorly and inferiorly to the glossopharyngeal nerve which runs in the third arch. It is the opinion of Ladd and Gross that a branchial fistula develops from the second branchial cleft since the common finding is a tract which runs between the internal carotid artery and the external carotid artery. It is also true that the internal opening of an incomplete internal fistula is at the base of the tonsil or posterior tonsillar pillar. This explanation as to the embryological etiology of branchial cyts corresponds with clinical findings in the great majority of cases when the tract can be followed to its termination.

Treatment.—Branchial cysts and fistulæ should be treated by complete surgical removal. Various methods have been advocated and no one method will fit every case. It is a simple and minor procedure if the sinus tract is short and does not extend deeply into the neck, however it becomes a major and a difficult procedure if it reaches as far as the pharynx. If the fistulous tract is short it can be excised through a small transverse incision along the normal crease of the skin. If it is found that the tract extends deeper into the neck, a second incision parallel to the first one, is made at a higher level close to the angle of the jaw (Fig. 119). This is the method first advocated by Bailey and is called the "stair step" incision. Vertical incisions made paralleling the anterior border of the sternocleidomastoid muscle give excellent exposure, however they leave ugly scars and for this reason are not recommended. The exception may be made for those cases with a badly scarred fistulous tract resulting from repeated infections or for those cases which have had previous attempts at removal. In this contingency, wide exposure is essential in order to avoid injury to the hypoglossal and accessory nerves and the important vital structures within the carotid sheath which might be closely adherent to the fistulous tract by adhesions.

Technique of "Stair Step" Method.—The first incision is made elliptically to encircle the external opening. It is extended transversely along a normal crease of the skin and long enough to permit easy dissection of the tract. It goes through the skin, platysma, and the subcutaneous tissue. The tract is now carefully freed from the overlying skin and underlying fascia covering the sternocleidomastoid muscle. Every effort should be made to avoid severing the tract. This can be done by using finger and scissors dissection while using only gentle traction on the fistuous tract during the process of its liberation. The dissection through this incision is carried upward and toward the angle of the jaw as far as possible. A second

incision is then made and the fistulous tract is delivered through this second incision as illustrated in Figure 119. This incision extends from the tip of the mastoid just beneath the angle of the jaw and parallels the first one. It is made sufficiently long to permit wide exposure of the structures within the carotid triangle. The skin flaps are dissected upward and downward and the surgeon now identifies

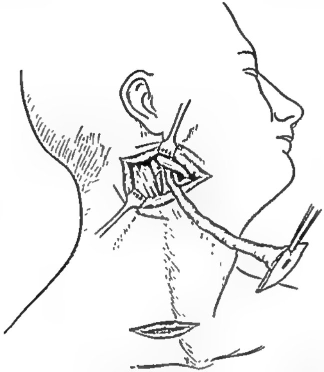


FIG. 119.—"Stair step" method of excising a branchial cyst. The first incision encircles the fistulous opening. The tract has been freed and the tract is delivered through a second incision higher up.

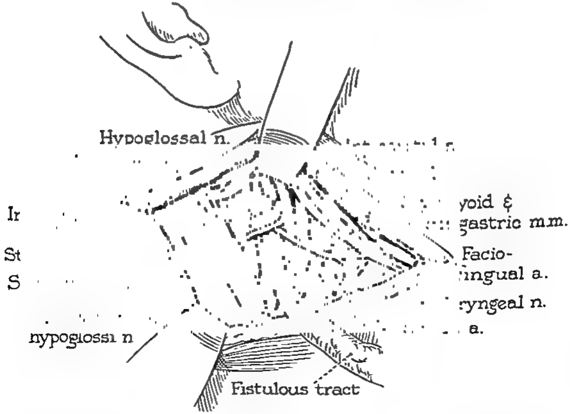


FIG. 120.—Illustrates the anatomical relationship of the fistulous tract. Note that it is found beneath the posterior belly of the digastric muscle and between the carotid vessels.

the various structures within the vicinity of the tract before proceeding with removal of the entire tract. Note that the tract courses laterally to the internal jugular vein and to both the internal and external carotid arteries until it turns into the pharyngeal wall when it passes between the two carotids. As illustrated in Figure 120, the tract lies beneath the posterior belly of the digastric muscle, medial to the sternocleidomastoid muscle and between the carotid arteries. The accessory nerve is found lateral to the tract and underneath the sternomastoid muscle. The hypoglossal nerve is in relation to the posterior belly of the digastric and the descending hypoglossal nerve lies on the internal carotid which is lateral to the tract just before the latter enters the pharynx.

The remaining portion of the fistulous tract is now dissected and removed to within a few millimeters of its termination at the base of the tonsil. When the pharyngeal wall is reached an assistant or the anesthetist places a finger in the mouth and pushes against the pharyngeal wall at the posterior pillar of the tonsil so that the surgeon can feel and determine the extent of the tract and thus complete the dissection and excision without puncturing the pharyngeal wall. When the dissection is completed, the tract is cut off within 2 or 3 millimeters of the pharynx. Some surgeons attempt to invaginate the remaining tract so that it will empty into the pharynx, however this is an unnecessary step and nothing is gained.

After all bleeding points have been controlled, the skin incision is closed in the same manner as for thyroidectomy. A small Penrose drain is inserted in the posterior angle of the upper incision and is removed in twenty-four to forty-eight hours.

The excision of a branchial cyst offers the same problem as the removal of a branchial fistula. The removal of the cyst without complete removal of the sinus leading to the pharynx is an incomplete operation and recurrence of symptoms is to be expected. In the case with infection and suppuration, the complete operation should be postponed until the infection has subsided. A suppurating sinus should be treated by wide excision of the external opening to increase drainage.

QUESTIONNAIRE

1. What other terms are used to describe a branchial cyst or fistula?
2. What is the origin of a branchial cyst?
3. Describe the three types of branchial cysts.
4. Give the clinical findings of these tumors.
5. What is the most prominent symptom of a branchial fistula?
6. Discuss the differential diagnosis.
7. Discuss the embryological etiology of branchial defects.
8. Discuss the etiology, symptoms and treatment of first branchial cysts.
9. Describe the "stair step" method of excising a branchial fistula.
10. Give the relationship of the fistulous tract as it proceeds to its termination in the pharyngeal wall.
11. How would you treat a suppurating infected branchial cyst or fistula?

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CHAPTER 19

SURGERY OF THE NECK—PHARYNGO-ESOPHAGEAL DIVERTICULA

By A. V. Partipilo

General Considerations.—Diverticula may occur at any portion of the esophagus, although in their order of frequency they are usually found at the following sites: (1) at the junction of the pharynx and the esophagus; (2) at the supradiaphragmatic portion; and (3) at the tracheo-bronchial bifurcation. Those occurring at the first two sites are considered as "false" diverticula because they are due to protrusion of the mucus membrane through the muscular coats of the esophagus. Thus, the sacs consist of mucosa only and are devoid of a muscular layer. Those occurring at the tracheo-bronchial bifurcation and elsewhere, the sacs are formed by the protrusion of the entire esophageal wall and are considered as "true" diverticula.

False diverticula result from pressure within the lumen of the gullet and are known as "pulsion" diverticula, whereas the true diverticula are the result of pressure from without and are known as "traction" diverticula. Anatomical and physiological factors present at the upper and lower ends of the esophagus are conducive to the development of pulsion type of diverticula. Those occurring elsewhere are due to traction from adhesions as a result of some inflammatory process adjacent to the esophagus. As a result of the attachment, the area is pulled outward in a funnel-like projection to form a V-shape deformity which extends from the esophagus. They are most frequently seen at the tracheal-bronchial bifurcation. They are often multiple and seldom reach the size of a pulsion type. The sac of a traction diverticulum is usually found at the anterior or lateral surface of the esophagus. The sac consisting of all layers of the esophagus is able to empty itself and for this reason surgery is seldom indicated. It is only in those cases when the sac cannot empty itself or when symptoms are persistent that surgery is considered. It must be emphasized that traction diverticula are not in the true sense of the word diverticula but rather dilatations, either localized or general, of the esophagus. They represent distortions of the esophagus and are not actual herniations.

The pharyngo-esophageal diverticulum, discussed in this chapter, is the most common type encountered. It occurs in the midline of the posterior wall at the junction of the pharynx and the esophagus (Fig. 121). From an anatomical point of view, the diverticulum should be termed pharyngeal rather than esophageal since the weak point is located in the triangular space bounded superiorly by the inferior constrictor muscle, laterally by the fibers of the latter muscle as they descend downward obliquely to the posterior wall of the esophagus to meet in the midline with the crico-pharyngeus muscle. The development of a diverticulum in this weak area is due to increased intrinsic pressure caused by an alteration or interference in the normal act of swallowing. During the second stage of degluti-

tion the crico-pharyngeus acts as a sphincter, while the oblique fibers of the inferior constrictor muscle relax and elevate the larynx. However, if the crico-pharyngeus fails to relax when the food is brought against it, the result is a propulsive force which is exerted against this weak spot of the pharynx. This force produces a tear in the musculature, thus initiating a pit in the mucosa. With each succeeding pulsion, the mucosal defect enlarges to finally produce a well-developed diver-

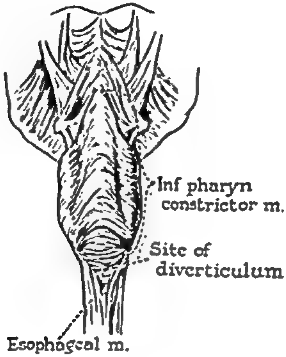

FIG. 121.—Common site for pharyngo-esophageal diverticulum.

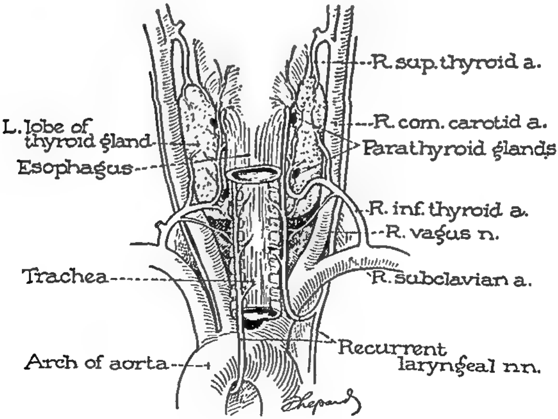

FIG. 122.—Illustrates the relationship of the recurrent laryngeal nerve to the thyroid gland and the esophagus.

ticulum. The mechanism is somewhat similar to the formation of a hernial sac in the inguinal canal. Because the esophagus is to the left of the midline in this region, the diverticulum is most frequently found to the left side of the neck.

Surgical Anatomy.—The esophagus is a muscular canal which extends from the pharynx to the stomach. The cervical portion begins at the level of the cricoid cartilage, descends along the front of the vertebral column, and ends underneath the clavicle to enter the mediastinum. It lies in loose areolar tissue in front of the prevertebral fascia.

Figure 122 illustrates the essential relationship of the esophagus. Note that the recurrent laryngeal nerves are respectively located on either side in the groove between the trachea and the esophagus and that they disappear beneath the inferior borders of the inferior constrictor muscles. The recurrent nerve must be found and isolated before attempting to free the hernial sac. Its point of entrance into the larynx is at the inferior horn of the thyroid cartilage which is a good landmark for the starting point in exposing the nerve. With a dissecting probe, the prevertebral fascia is pierced or "tweezed" just below the inferior horn until the white shining fibers of the nerve are seen. From this point on, the nerve is followed downward by cutting the fascia with short sweeps until the entire nerve is exposed. It is important to bear in mind that the right nerve is more often in front of the inferior thyroid artery and for this reason it is more apt to become injured while ligating the artery. Note also the relationship of the thyroid lobes which must be dislocated to expose the sac. The approach to the esophagus in this region requires the same anatomical knowledge as when performing a total thyroidectomy. Hence, the reader is referred to the chapter on Anatomy of the Neck for more detailed surgical anatomy of this area.

Preoperative Treatment.—In addition to the general supportive treatment, there are some specific preoperative conditions which must be corrected before the patient is brought to surgery. It is highly desirable to have the diverticulum empty of food, barium and other debris. Some patients have found by experience methods of emptying the sac voluntarily. It is important to realize that the pharynx and esophagus are grossly contaminated with virulent bacteria. As a prophylactic measure, penicillin and streptomycin should be given the forty-eight hours preceding surgery. An antero-posterior roentgenogram after a barium swallow is essential in order to locate the side to which the diverticulum has deviated. This should be done many days prior to surgery.

CONSIDERATION OF THE ONE-STAGE AND TWO-STAGE METHODS

The surgical treatment of esophageal diverticula, which were hitherto attended with a high mortality rate, is at the present time associated with relatively low mortality and morbidity rates. This has been due to a wider and better understanding of the anatomy of the region; to the use of antibiotics pre- and postoperatively; to improved anesthetic techniques and to refinement of operative technique.

Whereas in the past, because of the danger of spreading infection, the two-stage method was the procedure of choice, today it is being replaced by the one-stage method. This is especially recommended for the small diverticula and the two-stage should be reserved for the very large diverticula.

Technique One-stage Method

The patient is placed on the operating table in a position similar to that for a thyroidectomy operation, except that the neck is not bent backward. Hyperextension of the head and neck places a strain on the trachea and esophagus thus making the diverticulum less accessible. The head is turned to the opposite side of the diverticulum which is usually on the left. The technique described herein and the illustrations are for a right-side approach. This is done because the drawings were made during the repair of a diverticulum which was located on the right side. Regardless of the side of the neck involved, the anatomy and technique are the same. Anesthesia is administered through an endotracheal tube. The tube is provided with an inflated rubber cuff or balloon to prevent backward escape of gases in the pharynx.

FIG. 123.—The skin incision made along the anterior border of the sternocleidomastoid muscle and centered at the level of the anterior convexity of the cricoid cartilage.

The Incision.—The approach to the diverticulum may be through an anterior or posterior longitudinal incision along the sternocleidomastoid muscle or through a regular collar incision. We prefer the anterior longitudinal one, reserving the collar incision when cosmetic consideration is a dictating factor.

The incision is made on the indicated side along the anterior border of the sternocleidomastoid muscle. It is 3 to 4 inches long and is centered at the level of the anterior convexity of the cricoid cartilage. The neck of the sac lies just behind the inferior border of the posterior surface of the cricoid cartilage, a point which corresponds to the portion of the cartilage palpable anteriorly in the neck. An incision extending one and one-half to two inches above and below this level gives an adequate access to the neck of the sac; however if the sac is large and extends into the mediastinum, the lower extension of the incision should reach the sternal notch. Indeed, since diverticula usually occur in older patients in whom the cricoid has dropped to a lower level, the lower end of the incision will usually reach the sternal notch.

Exposure of the Sac.—The incision thus made passes through the skin, subcutaneous tissue and platysma muscle. Two cats'-paw retractors are placed on the lateral edge of the incision and by firm traction laterally with countertraction medially, a one-inch flap is raised in a line of cleavage between the platysma and the deep cervical fascia. A similar flap is raised medially. The fascia along the sternocleidomastoid muscle is incised and separated from the sternohyoid muscle

which is displaced medially. The lateral edge of the sternothyroid muscle is prolonged as a thin fascial sheath to the great vessels. This fascia is incised and the muscle retracted medially. The anterior belly of the omohyoid is cut and removed. As illustrated in Figure 124, two retractors are used to expose the thyroid gland and the structures within the carotid sheath.

The next step in the operation is to mobilize the thyroid gland in order to adequately exposed and free the diverticulum. This is accomplished by ligating and cutting the middle thyroid veins and if sufficient medial displacement of the gland cannot be obtained the inferior and superior vessels may likewise be cut and ligated. The gland is now either grasped with a Babcock forceps or held with a temporary traction suture passed through its substance and then rotated medially and anteriorly, thus exposing the trachea, the esophagus and the underlying hernial sac.

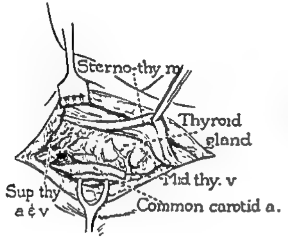

FIG. 124.

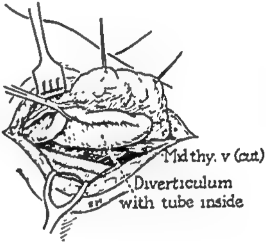

FIG. 125.

FIG. 124.—Two retractors are used to expose the thyroid gland and the structures within the carotid sheath.

FIG. 125.—The thyroid gland has been mobilized and the endotracheal tube or a large gastric lavage tube has been inserted into the diverticulum.

Before proceeding with mobilization of the sac, the anesthetist passes a large gastric lavage tube or an endotracheal tube into the esophagus. As the tube is pushed downward it will invariably enter the diverticulum. Gentle pressure upon the tube outlines the sac permitting a more defined and complete separation of the sac. This technique is an unpublished method of resecting an esophageal diverticulum by Lindon Seed* who has graciously consented to permit us to describe it in this chapter. We have found that with the use of the tube, as suggested by Seed, the entire extent of the sac can be accurately determined thereby greatly facilitating the delineation of its neck and preventing too tight closure.

With gentle traction of the thyroid gland and retraction of the sternocleidomastoid muscle, the prevertebral fascia is incised down to the mediastinum. This is an important and essential step in order to obtain some degree of rotation of the esophagus and exposure of the sac. Before proceeding with the dissection, the recurrent laryngeal nerve must be identified and protected. It can be easily identifield as it enters the larynx under the lower border of the inferior constrictor muscle just behind the inferior horn of the thyroid cartilage. Although the nerve is

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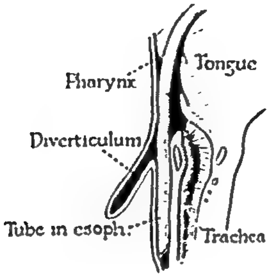

FIG. 126.—After the sac has been mobilized, the tube is directed into the lower esophagus.

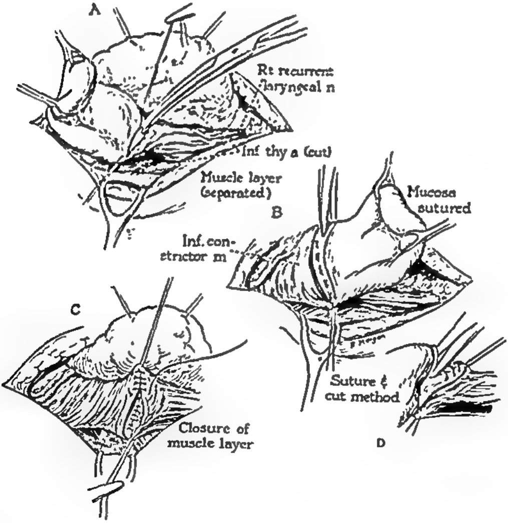

FIG. 127.—*A*, The sac has been mobilized and the m
the sac, in s
sac excised.
D, An alter

anteriorly placed in relation to the esophagus, nevertheless, one must tak account the possibility that the origin of the diverticulum may not alwa exactly in the midline and on the posterior surface of the esophagus.

As illustrated in Figure 125, the diverticulum with the indwelling tube is gr with a Babcock forceps and the sac is carefully separated from the esophag to the point where it becomes continuous with the esophagus. Actually tl lies in loose areolar tissue and can be freed even though it may extend to the astinum by applying successive rows of Babcok forceps as the sac is gently

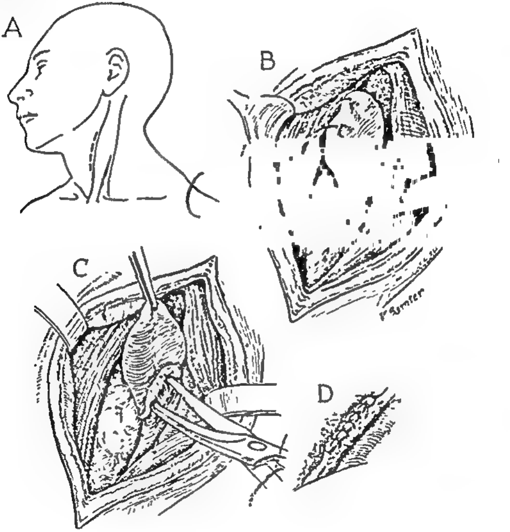

Fig. 128.—The essential steps in the two-stage method of resecting the diverticulum.

pulled upward. After the sac has been completely mobilized, the tube is dir into the lower portion of the esophagus as illustrated in Figure 126. With the in this position the sac is now definitely delineated and the chances for stri subsequent to its closure is materially lessened.

Resection of the Sac.—The sac of a diverticulum can be compared to th direct inguinal hernia whose internal opening or neck is not clear-cut thus mak difficult to determine the exact point for excising the sac. As illustrated in F 127*A*, a guy suture is placed along the neck of the sac at the point where it trudes between the inferior constrictor muscle and the esophagus. The cr cartilage along with the pharynx and esophagus is rotated medially, 180° if pos Another guy suture is placed in a similar position on the opposite side of the

With careful blunt dissection, Figure 127*A*, the muscular layer is freed above and below the diverticulum. The mucosa is now fully exposed and the sac can now be resected by any of the following methods: (1) the neck of the sac is clamped with an angiotribe forceps, the diverticulum is cut off and the base is sutured with a series of interrupted or continuous 00 intestinal catgut sutures; (2) a somewhat similar technique, except that no clamps are used (Fig. 127*B*) and (3) the neck of the sac is transected for a short distance, then sutured (Fig. 127*D*). The procedure of cut and suture is continued until the neck is completely excised and closed. The closure of the defect is now completed by inserting one or more series of interrupted fine silk sutures to the muscular layer (Fig. 127*C*).

The operation is completed by cutting the two guy sutures and the removal of the tube. If a Levine tube is desired, it may be introduced through the larger endotracheal tube before the latter is removed.

A Penrose drain is placed in the operative area. This should not be removed for at least one week when the danger of leakage has passed.

A few interrupted sutures approximate the sterno-hyoid muscle. The platysma and skin is then closed in the usual manner.

Technique of the Two-stage Method

The exposure and incision for the two-stage method of resecting a diverticulum is the same as for the one-stage method. Essentially the procedure consists of freeing the sac and suturing it to the sternocleidomastoid muscle as illustrated in Figure 128*B*. This must be done as high as possible to permit adequate emptying and excision of the sac. At the second stage, done in about ten days to two weeks, the sac is excised and closed as illustrated in Figure 128*C* and *D*.

QUESTIONNAIRE

1. Where are the most frequent sites for development of esophageal diverticula?
2. What is a "false" diverticulum?
3. What is meant by a "true" diverticulum?
4. What is the mechanism in the formation of the above types of hernia?
5. Define "pulsion" and "traction" diverticulum.
6. Where is the sac of a traction diverticulum found in relation to the position of the esophagus? And also of the pulsion diverticulum.
7. Are traction diverticula actual esophageal herniations? Explain.
8. Give the anatomical site of pharyngo-esophageal diverticulum?
9. Why should these be considered as pharyngeal rather than esophageal?
10. Discuss the method of development of these diverticula.
11. On which side of the neck are they most frequently found? Why?
12. Give the anatomy of the cervical esophagus.
13. How would you locate the recurrent laryngeal nerve?
14. Which is the preferred method of resecting a diverticulum, the one-stage or two-stage? Why?
15. Which incision would you use in exposing the hernia?
16. Describe the technique of the one-stage method.
17. What are the advantages in the use of large caliber tube in the esophagus while resecting the sac?
18. Describe different methods of closing the neck of the sac.
19. Describe the essential steps of the two-stage method.

CHAPTER 20

THE SALIVARY GLANDS

By A. V. Partipilo and S. W. Ondash

General Considerations.—Interest in surgery of the salivary glands has been stimulated by an expansion of the application of bolder cancer surgery to this field. This applies notably to the parotid gland which is being approached with less fear because surgeons are becoming more familiar with the anatomy of this long neglected area. A wider knowledge of the anatomy of the parotid region has led to more commonplace extirpation of the gland without irreparable damage to the facial nerve except in instances where total or segmental excision is indicated by its involvement in tumor. Even then, sacrifice of the nerve presents much less concern in the light of new and improved techniques which can be utilized in reestablishing its anatomic and physiologic continuity.

While efforts to achieve better results in the management of salivary gland tumors have failed to resolve all differences about their histogenesis and pathological classification, more unanimity of opinion has been reached regarding their surgical management. Present concepts of therapy have been evolved after critical consideration of the unfavorable clinical behavior of the so-called "mixed tumors" about which so many controversial problems have been raised. It is the clinical behavior of these tumors which has undoubtedly stimulated increasing curiosity as to their biological activity and has pressed the need for better pathological classification, more extensive surgical management and closer clinical follow-up. High recurrence of what were once believed to be inherently benign tumors, has particularly emphasized the need for extension of the scope of surgery directed toward therapy.

Numerous accounts in surgical literature dealing critically with the anatomical and embryological structure of the parotid gland, have provided a useful purpose. They have given important information on variations in the size of the gland, its position and most importantly, its relation to the facial nerve and its varied pattern of distribution. Accordingly, present day surgical approach to the gland is more generally following the basic tenets of good cancer surgery in that prompt, safe and more extensive excision follows clinical recognition of these tumors, the most benign of which carry malignant potential.

Histological and Embryological Considerations.—The salivary glands, arranged in pairs, consist of the parotid, sub-maxillary, sub-lingual and many smaller glands of the oral cavity. Parts of the larger glands may be displaced into surrounding tissues. The glands are of epithelial origin and develop from nodular invagination of the primitive oral epithelium into adjacent mesodermal tissue. The parotid glands are the first to develop and appear during the fourth week of embryonal life. Epithelial cords proliferate and branch in a tree-like fashion, thus forming the distally located alveoli. As central lumina appear in the distal accumulations of epithelial cells, they differentiate gradually in the characteristic cells of the salivary glands.

Microscopically, salivary glands are of tubulo-alveolar structure grouped in tubules, and lobules of the large glands form lobes. The lobules and lobes are supported by strands of loose connective tissue containing many blood vessels. The alveoli are composed of either serous or mucous cells or of a mixture of both. The so-called "myo-epithelial cells" are located between the secretory cells and the basement membrane as well as between the cells themselves. The intercalated ducts or isthmuses are zones of proliferation and have a simple low cuboidal epithelium while the larger striated tubules are lined with columnar epithelial cells. The secretory ducts have acidophilic pseudostratified cylindric cells and "myo-epithelial cells." The glandular elements are innervated by parasympathetic fibers. There are often islands of lymphoid tissue in the larger salivary glands namely, the parotid gland and there are often inclusions of salivary gland elements in the pre-auricular lymph nodes.

THE PAROTID GLAND

Anatomical Considerations.—The parotid gland, largest of the salivary glands, lies on the side of the face, below and in front of the ear, the mastoid process and the sternocleidomastoid muscle. It is contained in a recess which lies behind the ramus of the lower jaw and below the base of the cranium. The parotid region is bounded anteriorly by the ramus of the mandible, posteriorly by the mastoid process and anterior margin of the sternocleidomastoid muscle, and superiorly by the external auditory meatus and the zygomatic arch. The inferior margin of the recess extends to the angle of the mandible.

The gland is invested in a fibrous capsule which sends processes into its substance and divides it into lobes and lobules, differing in this respect, from the submaxillary gland which is loosely enveloped in its sheath and can easily be shelled from it. The fascial investment of the parotid is continuous with the deep cervical fascia. It provides a lining for the parotid recess and is united above to the periosteum over the acoustic meatus and posterior part of the glenoid fossa; medially it is connected to the styloid process, while below it joins the deep cervical fascia.

There is considerable controversy regarding the gross anatomy of the gland. Divergent opinion concerns the unilobar or bilobar structure of the gland and whether the facial nerve pierces the substance of the gland or lies in the tissue planes between the lobes.

Winsten and Ward, in a recent critical anatomical study, point to a unilobar configuration of the gland and indicate that the facial nerve branches are intimately enmeshed within the glandular substance. They discredit the predominant concept originated by McWhorter that the gland has two distinct lobes and that the nerve lies in the tissue planes between those lobes. They have demonstrated that the facial nerve appears in the face of the human embryo prior to the development of the parotid gland and that the gland grows out from the primitive oral cavity across the masseter muscle and around the ascending ramus of the mandible posteriorly to lie on the pharynx at the same time the gland grows between the branches of the nerve. Their study indicates that there is no demonstrable cleavage line between the gland and the nerve.

Beahrs, Martin, Lathrop and Tabah also share the opinion that portions of the gland lie superficial or deep to the nerve which pierces its substance shortly after leaving the stylomastoid foramen.

Davis *et al.*, in a recent study of 350 cervicofacial halves of the parotid gland,

CHAPTER 20

THE SALIVARY GLANDS

By A. V. Partipilo and S. W. Ondash

General Considerations.—Interest in surgery of the salivary glands has been stimulated by an expansion of the application of bolder cancer surgery to this field. This applies notably to the parotid gland which is being approached with less fear because surgeons are becoming more familiar with the anatomy of this long neglected area. A wider knowledge of the anatomy of the parotid region has led to more commonplace extirpation of the gland without irreparable damage to the facial nerve except in instances where total or segmental excision is indicated by its involvement in tumor. Even then, sacrifice of the nerve presents much less concern in the light of new and improved techniques which can be utilized in reestablishing its anatomic and physiologic continuity.

While efforts to achieve better results in the management of salivary gland tumors have failed to resolve all differences about their histogenesis and pathological classification, more unanimity of opinion has been reached regarding their surgical management. Present concepts of therapy have been evolved after critical consideration of the unfavorable clinical behavior of the so-called "mixed tumors" about which so many controversial problems have been raised. It is the clinical behavior of these tumors which has undoubtedly stimulated increasing curiosity as to their biological activity and has pressed the need for better pathological classification, more extensive surgical management and closer clinical follow-up. High recurrence of what were once believed to be inherently benign tumors, has particularly emphasized the need for extension of the scope of surgery directed toward therapy.

Numerous accounts in surgical literature dealing critically with the anatomical and embryological structure of the parotid gland, have provided a useful purpose. They have given important information on variations in the size of the gland, its position and most importantly, its relation to the facial nerve and its varied pattern *of distribution. Accordingly, present day surgical approach* to the gland is more generally following the basic tenets of good cancer surgery in that prompt, safe and more extensive excision follows clinical recognition of these tumors, the most benign of which carry malignant potential.

Histological and Embryological Considerations.—The salivary glands, arranged in pairs, consist of the parotid, sub-maxillary, sub-lingual and many smaller glands of the oral cavity. Parts of the larger glands may be displaced into surrounding tissues. The glands are of epithelial origin and develop from nodular invagination of the primitive oral epithelium into adjacent mesodermal tissue. The parotid glands are the first to develop and appear during the fourth week of embryonal life. Epithelial cords proliferate and branch in a tree-like fashion, thus forming the distally located alveoli. As central lumina appear in the distal accumulations of epithelial cells, they differentiate gradually in the characteristic cells of the salivary glands.

Microscopically, salivary glands are of tubulo-alveolar structure grouped in tubules, and lobules of the large glands form lobes. The lobules and lobes are supported by strands of loose connective tissue containing many blood vessels. The alveoli are composed of either serous or mucous cells or of a mixture of both. The so-called "myo-epithelial cells" are located between the secretory cells and the basement membrane as well as between the cells themselves. The intercalated ducts or isthmuses are zones of proliferation and have a simple low cuboidal epithelium while the larger striated tubules are lined with columnar epithelial cells. The secretory ducts have acidophilic pseudostratified cylindric cells and "myo-epithelial cells." The glandular elements are innervated by parasympathetic fibers. There are often islands of lymphoid tissue in the larger salivary glands namely, the parotid gland and there are often inclusions of salivary gland elements in the pre-auricular lymph nodes.

THE PAROTID GLAND

Anatomical Considerations.—The parotid gland, largest of the salivary glands, lies on the side of the face, below and in front of the ear, the mastoid process and the sternocleidomastoid muscle. It is contained in a recess which lies behind the ramus of the lower jaw and below the base of the cranium. The parotid region is bounded anteriorly by the ramus of the mandible, posteriorly by the mastoid process and anterior margin of the sternocleidomastoid muscle, and superiorly by the external auditory meatus and the zygomatic arch. The inferior margin of the recess extends to the angle of the mandible.

The gland is invested in a fibrous capsule which sends processes into its substance and divides it into lobes and lobules, differing in this respect, from the submaxillary gland which is loosely enveloped in its sheath and can easily be shelled from it. The fascial investment of the parotid is continuous with the deep cervical fascia. It provides a lining for the parotid recess and is united above to the periosteum over the acoustic meatus and posterior part of the glenoid fossa; medially it is connected to the styloid process, while below it joins the deep cervical fascia.

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Davis *et al.*, in a recent study of 350 cervicofacial halves of the parotid gland,

support the concept expressed by Bailey and reaffirmed by others that "although there are considerable variations in the disposition of the nerve within the gland, for practical purposes it may be stated that much more often than not, the facial nerve lies between a comparatively large superficial lobe and a variably sized deep lobe, the two being connected by an isthmus. It is this general anatomical concept that the operation of superficial lobectomy and complete parotidectomy are rendered practicable."

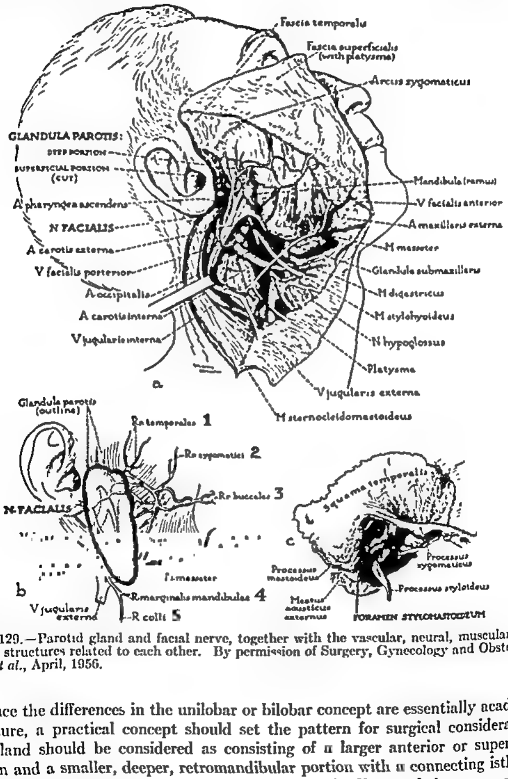


FIG. 129.—Parotid gland and facial nerve, together with the vascular, neural, muscular, and skeletal structures related to each other. By permission of Surgery, Gynecology and Obstetrics; Davis *et al.*, April, 1956.

Since the differences in the unilobar or bilobar concept are essentially academic in nature, a practical concept should set the pattern for surgical consideration. The gland should be considered as consisting of a larger anterior or superficial portion and a smaller, deeper, retromandibular portion with a connecting isthmus or isthmi of varying size. The lower portion winds itself around the ramus of the mandible to its medial surface where it enters the retromandibular space and forms the retromandibular element.

Operative experience also indicates that the facial nerve is intimately enmeshed in the gland and dissection fails to disclose simple cleavage planes between them. We prefer not to simplify the relationship of the nerve to the gland but rather to emphasize its intimate association with its substance. Any experience with dissection will indicate that it is difficult to demonstrate separate and distinct lobes divided by fascial envelopes with the nerve simply sandwiched between them. Instead, the nerve will be found to pass through parotid fascia and to lie directly in parotid parenchyma for most of its distance.

Fascia.—The fascia investing the parotid is derived from the enveloping fascia of the neck. The glandular investment is continuous with the deep cervical fascia. The outer layer, continuous with the fibrous sheath about the sternocleidomastoid

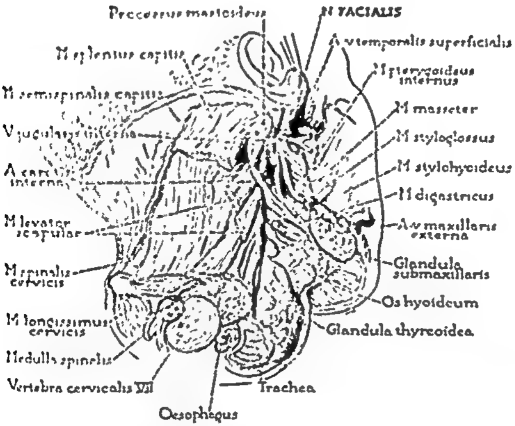
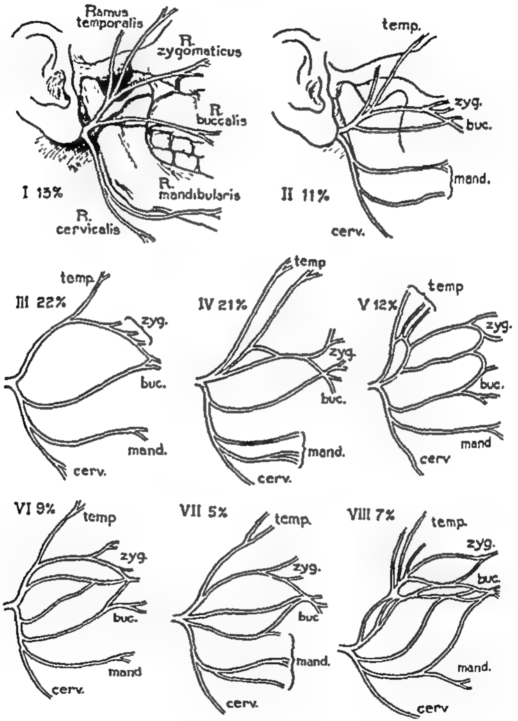


FIG. 130.—Structures which form, or are closely related to, the fossa for the parotid gland. The latter has been totally removed, the sternocleidomastoid muscle cut away and the great vessels transsected. By permission of Surg., Gynec., and Obst., Davis *et al.*, April, 1956.

and masseter muscles, is attached to the zygomatic arch. The deep layer, derived from the same sheath, runs deep to the parotid gland toward the pharynx. This dense sac of fascia encasing the gland is closed below but open above. There is a gap in the deep wall of the fascia between the styloid process and the internal pterygoid muscle through which the parotid space communicates with the connective tissue about the pharynx. A retropharyngeal abscess may, therefore, push the parotid outward. In some cases these abscesses have been evacuated into the parotid space.

Facial Nerve.—The anatomy of the facial nerve commands particular interest in that it holds the key to surgery of the parotid gland. A growing understanding of its course in relation to the gland has enabled a bolder surgical approach to tumors of the gland and serves as forceful argument to more radical procedures in

established or questioned malignancy. It is a mixed nerve and consists of two parts, a large motor, which supplies all the superficial muscles of the scalp, face and neck and some of the deeper ones; and a smaller sensory part which contains taste fibers for the anterior two-thirds of the tongue. It emerges from the brain at the inferior border of the pons, below the trigeminal nerve and medial to the



[illegible] (temporal and facial), [illegible]

acoustic nerve. After entering the internal acoustic foramen and passing through the canalis facialis in the petrous portion of the temporal bone, it emerges at the base of the skull by way of the stylo-mastoid foramen. It then passes anteriorly, laterally and slightly inferior for approximately one centimeter, then enters the posterior border of the gland. Thereafter, it courses through the parotid gland to supply the muscles of the face and with the exception of one or two small muscular rami, all branches arise after the nerve has reached the gland. Distribution of these rami is variable with the primary divisions remaining separate in some cases; in

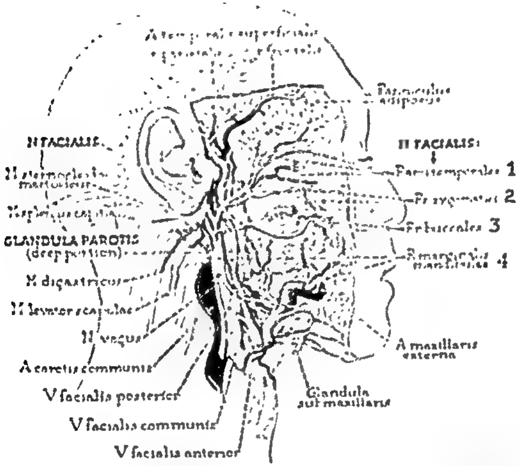


Fig. 132.—Facial nerve, deep leaf of the parotid gland, parotid duct, and related blood vessels. Illustrates the relation of the trunk of the facial nerve to the mastoid process and the anterior bor-

...Surg., Gynec. and Obst.

others, varying plexiform arrangements occur. Figure 131 demonstrates the variable distribution of the facial nerve as evolved in a study of one hundred cadaver dissections by McCormack *et al.* Chief types of branching encountered in 350 cervicofacial halves by Davis *et al.*, are represented by Figure 131, *I*, *II*, *III*, *V*, *VI* and *VIII*. Both McCormack and Davis, and their co-authors indicate that the point of bifurcation of the facial nerve lies posteriorly and slightly medial to the ramus of the mandible and superiorly two-thirds of the distance between the external angle of the mandible and the temporo-mandibular articulation. The two primary divisions are the temporo-facial and the cervico-facial with the former being consistently the larger. The principal branches arising from these two divisions are

the temporal, zygomatic, buccal, mandibular and cervical and they emerge at the anterior border of the gland.

Obviously, the preservation of the facial nerve during parotid surgery depends upon the recognition of the more common nerve patterns after division of the main nerve trunk and the awareness that many ramifications occur in the character of anastomoses between those two divisions. Only piecemeal dissection can assure recognition of these branches and their preservation during parotidectomy.

Blood Vessels.—Inconsistencies mark anatomic accounts of the blood vessels in and about the parotid gland. The most important ones entering and leaving the gland are the anterior and the posterior facial vein, the external carotid artery with its terminals and the superficial temporal and internal maxillary arteries. The anterior facial vein quite regularly courses along the margin of the mandible posterior to the branches of the facial nerve and then joins the posterior facial vein. The posterior facial vein is generally posterior to the gland or is covered by the superficial lobe of the parotid. Usually a large retrocondylar tributary of the posterior facial vein courses deep to the ramus of the mandible. Numerous venous channels occur between the two portions of the gland. The transverse facial artery and accompanying vein run forward beneath the superficial lobe just inferior to the zygomatic process. The external carotid artery courses deep within or beneath the gland in its superior and medial aspect. Numerous fine tributaries course between and supply the two portions of the gland.

Parotid Duct.—The parotid duct lies approximately two cms. below the zygomatic arch; it emerges from the most anterior part of the gland and runs forward on the masseter muscle to its anterior margin. At its origin it usually is superficial to the adjacent facial nerve branches. After coursing to the anterior margin of the masseter muscle, it passes medially, piercing the buccinator muscle obliquely to reach the mucous membrane of the cheek. It then opens into the mouth opposite the upper second molar tooth.

Lymph Nodes.—The parotid gland presents numerous nodes in relation to its surfaces and within its capsule. The anterior or pre-auricular group drain the temporal and frontal regions of the scalp, the outer portions of the lids and the outer ear. The parotid group scattered throughout the gland drain the upper and posterior part of the nasopharynx, the soft palate and the middle ear. Drainage may also take place directly to the mid-mandibular nodes or to the superficial nodes in relation to the external jugular vein.

HISTOPATHOLOGICAL CONSIDERATIONS

The most important pathological conditions involving the parotid gland are acute inflammations and tumors, with the latter being by far the most common.

Inflammation.—While inflammation of the parotid is relatively uncommon, it does occur. The inflammation may be suppurative or non-suppurative in character. The non-suppurative variety is known as "mumps" and terminates by resolution whereas the suppurative form results in purulent exudate and abscess formation. This form of acute suppurative parotitis results from infection which usually spreads along Stenson's duct. Such infection usually complicates calculous impaction of the duct and is manifested by reddening or pouting of the orifice of the duct at its emergence opposite the second upper molar tooth.

Tumors.—The study of parotid tumors has resulted in a wide variety of classi-

fications and considerable confusion has marked attempts to classify them. And yet, there is little question as to the importance of tissue diagnosis in the projected plan of treatment which may, in some instances, mandate sacrifice of the facial nerve and homolateral neck dissection. The presence of marked vairations in cellular pattern seen in any one tumor, the relatively slow growth of some of these tumors and the unpredictable habits of growth of some of them; all serve to indicate the vast challenge involved in a consideration of these tumors when first seen.

Some of these tumors are definitely benign, some are definitely malignant and the controversial group are referred to as "mixed" benign or malignant. In addition to characteristics of mixed cellular structure, these neoplasms also exhibit "mixed" growth patterns, benign in that they rarely metastasize but malignant in that they recur. The tumors vary from relatively acellular to cellular patterns of highly malignant neoplasms with the various stages of transition making exact classification difficult.

The surgeon must however, depend on a simple, workable classification from which he can adopt a satisfactory surgical approach.

Disregarding the connective tissue tumors such as lipomas, fibromas, fibrosarcomas as well as the angioendotheliomas, primary tumors of the parotid are all epithelial in origin. The following classification by Rawson *et al.*, is simple enough and a workable one since it provides a general guide for determining the type and extent of treatment for parotid neoplasms.

A. Benign epithelial tumors
   1. Mixed tumors
   2. Papillary cystadenoma lymphomatosum
   3. Sebaceous gland tumor
   4. Adenomas
      a. Ordinary adenoma
      b. Oncocytic adenoma

B. Malignant epithelial tumors
   1. Cylindroma
   2. Malignant papillary cystadenoma
   3. Adenocarcinoma
   4. Undifferentiated carcinoma
   5. Mixed carcinoma (mucoepidermoid carcinoma).

C. Miscellaneous tumors
   1. Benign
      Cyst
      Lipoma
      Neurofibroma
      Lymphocytic tumor
      Hemangioendothelioma

   2. Malignant
      Fibrosarcoma
      Neurosarcoma
      Melanoma
      Lymphosarcoma
      Hodgkin's

Mixed Tumors.—Any mention of the parotid gland brings concomitant reference to mixed tumors which comprise, by far, the largest number of clinical problems involving the gland. While inherently benign, recurrence after surgical extirpation has, in the past, varied from 20 to 45 per cent. Growth is characteristically slow for many years but the tumor may take on rapid growth at any time and invade surrounding tissues. On rare occasion, the mixed tumor may develop into a carcinoma. Rawson places this incidence rate at 5 per cent. Lymph nodes are seldom involved unless the tumor is interfered with but unsuccessful removal may result in subsequent invasion of the cervical nodes.

The "mixed tumor" designate was first made in 1874 but Boyd suggests that complex adenoma is a better name. Willis prefers to refer to the tumor as a pleomorphic adenoma. The designation "mixed" is unobjectionable as long as it is clearly understood that the tumor is not of mixed epithelial and connective tissue origin but of epithelial tissue alone. The great majority are benign but malignant mixed tumors do occur in approximately 6 per cent of cases and these may be difficult to distinguish from primary adeno-carcinoma. The multicentric origin of some of the mixed tumors provides reasonable explanation for some recurrences and adds emphasis to the need for broadening the scope of surgical excision.

Microscopically, mixed tumors are a complex structure composed of epithelial elements in the form of alveoli, cell strands, or diffuse masses interspersed in mesoblastic tissue made up of variable amounts of cartilage, hyaline or myxoid tissue or mucous tissue. The tumor rarely demonstrates any uniformity of pattern with varied elements being present in different areas of the tumor.

According to Ward and Hendrick, some tumors present columns of epithelial cells which are interspersed in mucoid connective tissue or cartilaginous tissue. The epithelium displays a great variability from adenomatous alveoli and gland acini to narrow strands or masses of flat compact epithelium of the basilar type, and small alveoli lined with cuboidal cells, which may be flattened so as to resemble lymph spaces lined with epithelium. Nests of squamous cells may appear in many tumors and some show pearl formation; whereas, in other sections glandular acini predominate with rather distinct adenomatous tissue.

Basal-like lesions with connective tissue or hyaline stroma are observed in a small group of tumors and may be seen near the surface or in the capsule of larger growths and are frequently associated with adenocystic-like structures. The cells in such tumors are small, cubical or spindle with hyperchromatic nuclei, and are arranged in narrow cords that anastomose with one another, or may form thin, flat strands. A muco-epidermoid variant is described by Foote and Becker. It arises from the duct epithelium and has a characteristic histological structure composed of two elements, squamous cells and tumor cells that produce mucin. The latter may be the origin of cysts. The malignant potentiality depends on the more cellular element which may break through the capsule and invade contiguous structures. These secondary growths are more definitely cellular and malignant.

Malignant Epithelial Tumors.—The following forms of malignant epithelial tumors are fairly generally accepted. Brief reference is made to their general pattern and clinical progression. Grossly, they are characterized by their lack of encapsulation, firmness, and infiltrative nature. All are best treated by radical excision of the gland.

Malignant Mixed Tumors.—These occur in approximately 6 per cent of cases and are difficult to distinguish from primary adenocarcinoma. The histological

structure may be adenocarcinoma, epidermoid carcinoma or spindle cell carcinoma. Involvement of lymph glands occurs in approximately 15 per cent of cases and as high as 40 per cent in the recurrent forms.

Cylindroma.—The cylindroma is a slow growing tumor in which local recurrence or lymph node involvement may occur over a period of years (Kirklin). Perineural lymph spread occurs in approximately 50 per cent of cases and the five-year cure is only 20 per cent (Foote and Frazell). Some consider it to be a form of adenocarcinoma but it is a distinctive tumor composed of cylindroid cords of cells containing a basophilic mucoid material. The tumor has shown a moderate degree of radiosensitivity initially but recurrence is the rule.

Malignant Papillary Cystadenoma.—This is the malignant variant of the papillary cystadenoma. The histologic picture shows considerable similarity with the exception of the malignant character of the epithelial cells lining the papillary projections. The tumor is locally invasive.

Adenocarcinoma.—Among the primary malignant tumors of the salivary glands, the adenocarcinoma deserves particular attention because of its great variety of patterns, infiltrating growth, frequent recurrence after removal and the tendency to metastasize. Dockerty and Mayo separated this neoplasm of the submaxillary salivary glands from the mixed tumors, with which it has been confused. Because of its resemblance to the so-called cylindroma, they named it "adeno-carcinoma of the cylindroma type."

The adenocarcinoma occurs as commonly in the submaxillary gland as the parotid. It grows slowly but relentlessly. Local recurrence or lymph node involvement may occur over a period of many years (Kirklin).

Muco-epidermoid Tumor.—Muco-epidermoid tumors of the salivary glands were recently described by Stewart, Foote and Becker. The authors claim that a little more than 5 per cent of all salivary gland tumors are of this type. The growth is solid or cystic, encapsulated or non-encapsulated, and originates from cells of the larger and intermediate ducts. The "mucus-forming epithelioma" of the salivary glands described by Skorpil, bears a definite resemblance to this tumor. It is characterized by abnormal differentiation into mucous cells and by epidermoid metaplasia. All of them are now considered to be malignant. The undifferentiated epidermoid carcinoma of the salivary glands, chiefly of the parotid, is rapidly growing and highly malignant.

SURGICAL CONSIDERATIONS

Opinion as to the surgical management of parotid gland tumors varies and has ranged from treatment by simple enucleation to total removal of the gland. Advocates of simple excision, except in the case of the very small benign lesion, are becoming few indeed, with the growing conviction that the tumor recurrence rate is high even in those cases where benignancy was well established. There is a definite trend toward superficial parotidectomy for any benign tumor regardless of size. H. Mason Morfit calls attention to the fact, that as the natural history of these growths is studied in greater detail, a satisfactory explanation of the high recurrence rate is provided. It serves to emphasize that the closer the surgeon approaches the ideal of complete removal of the proven malignant or benign tumor the more certain will he be of curing his patient.

In the past, the operative management of tumors of the parotid has been lim-

ited by a primary consideration of preservation of the facial nerve. Conservatism marked efforts to achieve this and inadequate excision, more often than not, resulted in recurrence. Indeed a number of tumors occurring in the retro-mandibular area, once considered anatomically inaccessible, were declared inoperable. Improved techniques in the approach to the gland have evolved a more rational approach to the problem and a more effective tumor therapy now appears imminent.

The surgical problem is that of completely excising the neoplasm and avoiding unnecessary injury to the facial nerve. In the interest of good cancer surgery, however and where there is invasion of tumor into the nerve, its sacrifice is of secondary importance. Today, newly accepted techniques of preserving the nerve have led to virtual abandonment of simple enucleations and limited excisions in favor of sub-total or total parotidectomy. Brintnall *et al.*, recommend total parotidectomy as a routine measure for all parotid tumors, including the benign variety in the superficial portion of the gland. They are reluctant to leave any parotid tissue in view of subsequent clinical behavior and feel that total excision of the gland carries no additional hazard. Others, notably Janes and Kergin of Toronto, Canada, feel that it is unnecessary to sacrifice the entire gland in cases of benign mixed tumor provided the tumor is excised with a substantial amount of normal gland. It is now generally agreed that nothing short of superficial parotidectomy will suffice even in the benign tumor save in the occasional instance where it is very small and well encapsulated.

The technical approach must fulfill the surgical principle of adequate exposure and identification of vital structures, the excision of the tumor and tumor potential, and the preservation of the facial nerve where it is not invaded by tumor tissue. *The extent of the removal of the gland must follow the established criteria of cancer* surgery elsewhere. The importance of biopsy of any tumor of the gland cannot be overemphasized. The biopsy should be excisional in character and aspiration biopsy is mentioned only to be discouraged. Aspiration biopsy, once popular, was used on the theory that a returned diagnosis of mixed tumor would spare more extensive surgery and possible nerve injury. New respect for the nature of mixed tumors and improved technique in surgical approach have popularized the belief that, other than occasional simple excision in the case of very small benign tumors, superficial parotidectomy is the minimal procedure to be employed in management.

The *unpredictable clinical behavior of any tumor* of the parotid makes histologic diagnosis imperative as a determinant of adequate treatment. If the frozen section proves indeterminate of malignancy, the wound should be closed and the tissue submitted to close paraffin study. Questioned reliability on the frozen section mandates the need for properly prepared and studied sections since the subsequent treatment may require complete excision with or without radical neck dissection. The surgeon therefore, must be prepared to interpret the histopathologic diagnosis in terms of prognostic implications and plan his approach accordingly. Guided by such consideration the extent of surgery will be one of the following: (*a*) extracapsular excision of the tumor; (*b*) superficial parotidectomy; (*c*) total parotidectomy or, (*d*) total parotidectomy combined with homolateral radical neck dissection.

TECHNIQUE OF PAROTIDECTOMY

Under Pentothal, nitrous, lorfan-nisentil intra-tracheal anesthesia with anectine utilized for the tubing process, the patient's head is turned to the contralateral

side and the operative region is draped to expose an area bounded above by the superior border of the ear, below by the clavicle, behind by the mastoid process and trapezius muscle and anteriorly by the midline of the neck and the angle of the mouth.

In small superficial tumors, the gland is exposed by a vertical incision anterior to the tragus of the ear. The skin and subcutaneous tissue are then divided and the tumor fully exposed. If the tumor is one centimeter or less in diameter and protrudes from the periphery of the gland it is widely excised and referred for frozen section. If the tumor is proven benign, the wound is then closed in conventional fashion utilizing interrupted sutures of fine silk or cotton. Where a very small tumor is located at the superior and medial portion of the parotid region, a horizontal incision can be utilized for what will presumably be surgery of extracapsular excision alone.

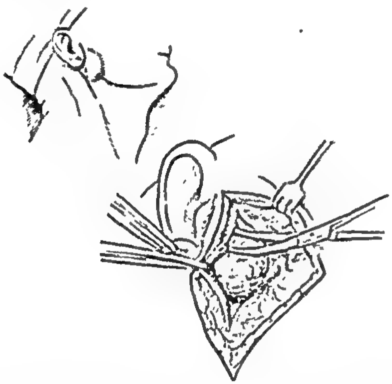


FIG. 133.—Incision is made anterior to the tragus of the ear. If tumor is large, it is converted into a Y. Lower diagram illustrates the reflection of the deep cervical fascia from the parotid gland and tumor.

Superficial Parotidectomy.—If the tumor exceeds one centimeter in diameter or projects into the deep portion of the gland and even though it is classed as benign, preparations are made for superficial parotidectomy. The original vertical incision in front of the tragus of the ear is prolonged inferiorly to a finger breadth below the angle of the mandible and then carried forward to the posterior border of the submandibular gland. The vertical component of the incision thus passes through the natural longitudinal crease in front of the external ear while the horizontal element lies under the ramus of the mandible. If the tumor is large this incision can be converted into a Y by placing another incision along the posterior aspect of the ear lobe. This procedure is particularly helpful in total parotidectomy where better access is gained to the retromandibular portion of the gland.

The ear is then kept in upward retraction by utilizing an anchoring suture drawing the lobule to the helix or superior portion of the appendage. The skin

flaps are then developed by sharp dissection along the length of the wound with the anterior or cheek flap being dissected forward thus exposing the central portion of the gland. The posterior flap is dissected sharply toward the mastoid bone and *adjacent sternocleidomastoid muscle thus exposing the tail of the gland.* Careful attention must be directed to hemostasis during the development of the skin flaps since brisk oozing may make identification of dissection planes difficult. The dissection is now carried deep in the zone behind the gland and close to the cartilaginous ear canal. Exposure of the upper border of the posterior border of the digastric muscle and its attachment to the mastoid process serves as an important landmark. By dissecting downward along the anterior border of the mastoid *process above the posterior belly of the digastric muscle, the main trunk of the nerve*

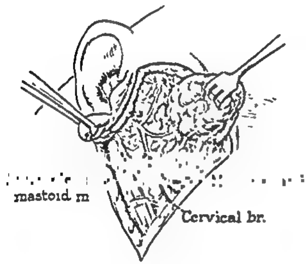


FIG. 131.

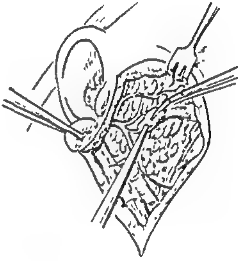


FIG. 135.

FIG. 131.—The superficial lobe of the parotid gland has been dissected without injury to the facial nerve branches.

FIG 135 —The posterior lobe of the parotid gland is removed by "piecemeal" dissection.

will be readily exposed. The nerve will be encountered at a depth of about one to one-and-a-half centimeters from the external surface of the mastoid process and one to two centimeters medial to the lateral surface of the tip of the mastoid. A V-shaped sulcus is found between the broad blunt anterior margin of the mastoid process and the fingernail like bony ridge at the antero-inferior margin of the external auditory meatus of the skull. At the sulcus, the nerve is but a few mms. from the depth of the dissection. I: [illegible] mastoid foramen is blocked, the lateral por [illegible]

Once the main trunk of the nerve is demonstrated, the gland is carefully dissected off under direct vision with the dissection following the two major branches of the nerve, *viz.*, the cervico-facial and the temporo-facial divisions. Occasionally, this main bifurcation occurs before the nerve enters the gland parenchyma. Each *of these two principal divisions divides into varied patterns* which are enmeshed between the superficial and deep portions of the gland. *Curved hemostats* are now used to good advantage in spread dissection superficial to the nerve thus liberating the superficial portion from the plexus of nerves lying beneath. The soft-sponge-

like consistency of the parotid tissue facilitates its peeling off from the firm taut nerve structure with which it is intimately associated. It will readily separate from the nerve with the aid of forward traction of the liberated gland substance.

As the dissection is carried medially, Stensen's duct, located at the anteromedial margin of the superficial portion is ligated and divided and the excision of the superficial portion is thus completed. Numerous bleeding points encountered during the course of dissection are ligatured with 0000 chromic catgut or 0000 silk or cotton.

Total Parotidectomy.- If the tumor is recurrent, its malignancy indeterminate or definitely established, or if the tumor extends to the deep portion of the gland, total parotidectomy is completed by "piecemeal" removal of the deep portion of the gland. Nerve hooks are conveniently used at this point to elevate the nerve filaments from the gland. The upper portion of the external carotid artery and the posterior facial vein groove the medial surface of the parotid deep to the facial nerve. These

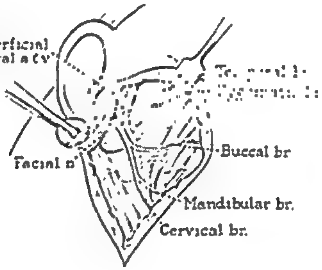


FIG. 136.—The complete dissection of the parotid gland.

are doubly ligated and transected at the inferior border of the gland. Hemostasis is achieved and the wound is then irrigated thoroughly with saline. The wound is closed with interrupted silk or cotton sutures after drainage is established with a Penrose drain through a small stab wound posterior to the incision in a dependent location beneath the angle of the mandible.

If there is gross evidence of invasion of the facial nerve or the lymph glands are involved in tumor, a radical *en bloc* removal of the gland and standard neck dissection is performed in conformity with good cancer surgery. A vertical incision dropped from the mid-point of the submandibular component of the original incision will provide adequate exposure for this procedure. A supraclavicular horizontal extension may sometimes be necessary to better facilitate the dissection.

It appears that the only objection to the aforementioned approach would be in instances where the tumor is so located that it would make dissection of the main trunk difficult or impossible without cutting into tumor tissue. Under such circumstances it would be impossible to dislodge a parotid tumor and expose the facial nerve without entering the tumor itself and thereby not act in accord with conventional tumor surgery. It would be well then, to consider another approach namely, one in which one of the peripheral branches of the nerve is identified and

dissection is pursued backward from that point to the more major ramifications. This blunt dissection is carried out gently and painstakingly after identification and division of the parotid duct.

It should be stressed that there is no absolute landmark for identifying a peripheral landmark nor is one branch more uniformly accessible than another. Since there are fewer blood vessels along the superior border of the parotid and since there is less fat and underlying tissues are firmer, the temporal or zygomatic ramus is the first to be identified in the course of such dissection. The buccal and occasionally, the zygomatic branches lie near the parotid duct and are readily identified. The dissection must be done carefully, gently and with strict attention to hemostasis. When the isthmus is reached the bifurcation of the main trunk into the cervico-facial and the temporo-facial divisions can be visualized. After removal of the superficial portion the deep portion is removed by ligating and transecting the external carotid artery and the posterior facial vein at the inferior border of the deep prolongation of the gland. The superficial temporal artery and vein are ligated and sectioned at the superior border and the internal maxillary artery is treated similarly at the mid-portion of the anterior border of the deep portion of the gland.

Surgery for Parotid Abscess.—A parotid abscess may be drained through an incision (Blair) which commences about an inch anterior to the ear and is carried downward behind and below the angle of the jaw. This is deepened through the capsule of the gland, and the parenchyma can then be opened by blunt dissection. The deep part of the gland may be drained by lifting the lower pole forward. It may be necessary to drain the space between the masseter and the superficial lobe of the gland, and this too can be accomplished through the same incision. A horizontal incision for the drainage of such an abscess may also be utilized.

Role of Irradiation in Tumor Therapy.—Radiotherapy is seldom, if ever, indicated therapeutically or post-operatively in the management of parotid tumors. Since the vast majority of tumors of the salivary glands are radio-resistant, and since radiation therapy distorts the histologic picture, radiotherapy should not be used prior to diagnosis. Mixed cell tumors are characteristically highly resistant to roentgen therapy. If, however, it has been impossible to remove all of the tumor or if recurrence is associated with poor risk status for secondary operation, external radiation may prove of value. Cylindromas are especially sensitive to irradiation. *Since they invade early and spread along the nerve roots, often reaching* the cranial cavity, irradiation over a wide area should follow excision of the gland involved in such tumor. Palliation can be achieved by repeated radiotherapy in the event of recurrence in some instances.

THE SUBMAXILLARY GLAND

Anatomical Considerations.—The submaxillary glands are next in size to the parotid glands and resemble them in lobulation and color. Each is located largely in the submaxillary triangle under cover of the mandible on the respective sides of the neck. In each gland two portions may be identified, a somewhat superficial larger element and a smaller part projecting from the deep surface of the body of the gland. The superficial portion presents a convex surface which projects below the mandible in the submaxillary triangle but it frequently extends beyond the limits of that space and overlaps the digastric muscle. The body of the gland is wedged upwards between the medial surface of the mandible and mylohyoid and

the hyo-glossus muscles. It thus presents two surfaces, a lateral which is in contact with the submaxillary fossa of the mandible and a medial, related to the mylohyoid, hyo-glossus, the posterior belly of the digastric and the stylo-hyoid muscles. The deep process passes around the posterior free margin of the mylo-hyoid muscle and comes to lie between the mylo-hyoid and hyo-glossus muscles.

The external maxillary artery lies embedded in a groove in the superior and posterior part of the gland. The gland is enclosed in a delicate capsule of connective tissue derived from the deep cervical fascia.

There are several lymph glands disposed about the submaxillary gland; the greater number lie immediately under the deep cervical fascia and the remainder between the gland and the mylohyoid muscle. More specifically, the midmandibular nodes are commonly considered as belonging to the submaxillary group. There are also a variable number of other lymph nodes in the submaxillary triangle. The submaxillary nodes complete the primary drainage of the lips, cheeks, lower gingiva and anterior parts of the tongue and in turn drain directly into the internal jugular chain, from the subparotid node to the omohyoid node but chiefly to the bifurcation nodes.

The hypoglossal nerve enters the submaxillary space between the hypoglossal muscle and the posterior belly of the digastric muscle and runs through the greater portion of the suprahyoid space before entering the sublingual compartment through the cleft between the hyoglossal and mylohyoid muscles. The lingual nerve lies at a much higher level on the lateral surface of the hyoglossal muscle and it supplies a branch to the submaxillary gland which must be severed in suprahyoid dissection before the gland can be mobilized in the process of removal.

Histopathological Considerations.—Like the parotid gland, the submaxillary gland can be involved in inflammation or tumor. True neoplastic enlargement of this gland is relatively uncommon. Tumefaction of the gland due to obstruction of Wharton's duct with or without associated inflammation is quite common. Pain and swelling provide distinctive symptoms of ductal obstruction and severity of these symptoms vary with the degree of obstruction and the amount of salivary secretion. Obstruction of the duct is usually due to calculi but nearby tumors can and do provide varying degrees of obstruction. *X*-ray examination is often helpful in detection of calculi and in some cases sialography is of value. Submaxillary abscesses arise from involvement incident to infections in the buccal cavity, teeth, tongue, gums or pharynx. The acute infection known as Ludwig's angina begins in the sublingual region but rapidly involves the submaxillary region.

Tumors occur infrequently but when they do they are similar to tumors of the parotid gland with the "mixed" tumor being most common. They have been discussed in the section dealing with Tumors of the Parotid Gland.

Surgery of the Submaxillary Gland.— *Ductal Obstruction.*—Treatment of ductal obstruction by calculus is achieved by removal after ampullary dilatation. When this is impracticable, an incision placed over the stone and parallel to the duct can be made with relative ease. Presence of multiple calculi in the substance of the gland with repeated infection and fibrosis mandate surgical excision of the entire gland. Abscess of the gland is treated by simple horizontal incision over the area of maximal fluctuation.

Excision of Submaxillary Gland.—Removal of the gland for chronic calculous fibrosis or tumor is achieved by placing a transverse incision approximately 3 to 4 centimeters below and behind the angle of the jaw. Progress is continued through

the platysma with a reflection of the skin and platysma upward thus avoiding injury to the mandibular branch of the facial nerve which supplies the muscles of the lower lip. The gland is easily exposed and then dissected from below and separated from the cellular tissue behind forward. The inferior border of the gland often extends below the great horn of the hyoid bone. The posterior extremity of the gland presents intimate and important relations with the parotid gland, facial artery, and thyroid, and hypoglossal and facial trunks of the common facial vein. The facial artery is deep in position and is found on the posterior surface of the gland. The facial veins, however course superficially over the submaxillary gland just beneath the fascial covering and are easily identified and ligated. The duct is dissected under the genio-hyoid muscle and divided and the excision at the gland easily achieved.

When frank carcinoma of the gland is established by biopsy or grossly evident from adherence to surrounding structures with invasion to adjacent lymph nodes, *en bloc* dissection should be carried out in conformity with the principles of good cancer surgery.

QUESTIONNAIRE

1. Discuss the embryology of the salivary glands.
2. Give the microscopic appearance of these glands.
3. Describe the topographic anatomy of the parotid gland.
4. Is the parotid gland unilobar or bilobar? Discuss.
5. What is the anatomical relationship between the facial nerve and the parotid gland?
6. What is the origin of the investing fascia of the gland?
7. Give the origin, distribution and function of the facial nerve.
8. At what point does it emerge from the cranium?
9. Describe the various manners in which the facial nerve may divide.
10. Describe the relationship between the various blood vessels to the facial nerve and the parotid gland.
11. Describe the course of the parotid duct.
12. Discuss the lymph drainage of the parotid gland.
13. [illegible]
14. [illegible]
15. [illegible]
16. [illegible]
17. [illegible]
18. [illegible] the parotid gland.
19. How would you treat them?
20. Discuss malignant papillary cystadenomas of the parotid gland.
21. Discuss the characteristics of adenocarcinoma of the parotid gland.
22. Discuss the surgical management of parotid gland tumors.
23. Describe the technique for parotidectomy: partial and complete operation.
24. How would you treat a parotid abscess?
25. Discuss the role of radiotherapy in parotid tumors.
26. Describe the anatomy of the submaxillary gland.
27. Describe the technique for excision of this gland.

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Many of the subcutaneous veins over the breast are more noticeable during pregnancy or lactation. Considerable significance has been claimed for their appearance under infra-red photographic study and certain patterns of the veins have been stated to have diagnostic importance. The veins draining the breast follow the arteries.

NERVES

No nerves of surgical importance go to the breast. The nerves of importance to the surgeon lie deep and lateral to it and supply the serratus anterior and the latissimus dorsi muscles. These are the long thoracic (nerve of Bell) and thoracodorsal nerves. The former lies on the anterior chest wall, making its entrance from behind the axillary vein at the apex of the axilla, descending vertically. The thoracodorsal nerve likewise descends from behind the axillary vessels, accompanies the subscapular artery along the axillary margin of the subscapularis muscle, and ends in the latissimus dorsi (*see* Fig. 137).

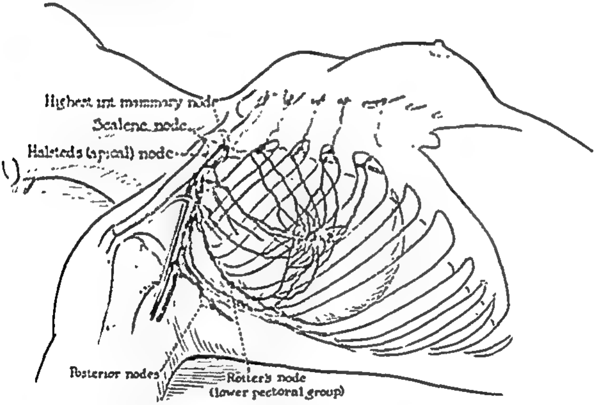

FIG. 140.—Lymphatic drainage of the breast.

LYMPHATICS

The lymphatic circulation of the breasts is a complex system of great surgical importance. The skin over the breasts contains its proper lymph vessels which communicate with each other and with those of the breasts, particularly in the subareolar plexus of Sappey which receives tributaries from the nipple and areola and from vessels around the lobules. The subareolar plexus drains into the axillary nodes by at least two large vessels which enter the anterior pectoral group of nodes, from which lymph may pass to the central axillary and subclavian nodes. The anterior pectoral nodes extend lowest, lying in the region of the lateral thoracic blood vessels. The central nodes are mid-axillary and the subclavian (Halsted's)

node is in the very apex of the axillary space. Lymph from the medial portion of the breast may drain directly into the nodes along the internal mammary artery and thence to the mediastinum, to similar nodes on the opposite side of the sternum and to the scalene node. Communications may extend from the subareolar plexus to the internal mammary nodes, and to the opposite breast. Lymph from the lowest part of the breast may pass through channels leading medially and inferiorly into collecting trunks which pass retrosternally into the mediastinum or through the abdominal wall to join vessels in the diaphragm. Lymph vessels penetrate the pectoralis major to end in an interpectoral group of nodes (Rotter's nodes). A small group of nodes along the subscapular vessels receives lymph from the lower outer part of the breast. The more important lymphatic pathways are indicated in Figure 140.

PHYSIOLOGY

Clinical Considerations.—The changes which take place in the breasts during adolescence, the menstrual cycle, pregnancy and lactation result from the interaction of various hormones. Ductal myoepithelium is stimulated to growth by estrogens, lobular structures by progesterone, and both are subject to the regulatory action of the pituitary hormone prolactin. Prolactin, adrenal and thyroid hormones, as well as placental estrogens, are involved in the mechanism of lactation. The ability of the liver to conjugate estrogens has its effect on hormonal balance. Clinically observable variations in the degree of response to hormone stimulation may result not only from hormonal imbalance, but from variability in the capacity of the breasts to react (unilateral hypertrophy, gynecomastia). The normal response of the breasts to periodic estrogen stimulation is by growth of ducts in length and caliber, budding of ducts, and hyperplasia of both duct epithelial and periductal connective tissue with retention of water in the latter. The extent and degree of the subsequent cyclic involution varies in different parts of the breasts, so that abnormality in either hyperplasia or involution or both may cause considerable variability in the type and location of the resulting dysplasia. The latter is most often clinically evident as nodularity or "lumpiness," usually tender, especially during the immediate premenstrual days. In the cases of young women the lumpiness is due mainly to overgrowth of fibrous connective tissue elements, while in those of older women hyperplasia of ductal epithelium and distention of duct structures are the more prominent changes.

All degrees of hypo- and hyper-involution may be seen in lobular structures at all ages. When women whose breasts have long been the sites of tender lumpiness approach menopausal age, the nodules may vary remarkably in size and consistency due to the formation of variable-sized clusters of tiny cysts. The generally fibrotic character of such breasts, however, restricts the enlargement of small cysts so that few become clinically appreciable lumps. Atrophy of breast glandular substance proceeds at an accelerated pace after the menopause. Lumpiness disappears gradually as the gland is replaced by fat, but some breast tissue capable of responding to stimulation by estrogens remains even in the breasts of old women. Further discussion of these functional changes in the breasts will be found under the heading Chronic Cystic Mastitis.

INFLAMMATORY LESIONS

Inflammatory lesions of the breasts are less commonly encountered since the advent of antibiotics but occur both as complications of lactation and in the non-

puerperal state. Swelling, redness, heat and painful tenderness are characteristic. Fever is usually present with acute inflammation, the axillary nodes may be enlarged and tender, and the overlying skin is often edematous ("peau d'orange" appearance) and adherent to underlying breast tissue.

Figure 141 indicates the possible locations of abscesses.

Acute lactation mastitis is treated with antibiotics, ice bags, cessation of nursing and snug support. If suppuration occurs, incision and drainage of the abscess is the treatment of choice. Incisions into the breasts should be made radially, parallel to the course of the ducts, but retromammary abscess can best be drained through an incision in the inframammary crease. Breast abscesses, when extensive, are often multilocular, a situation discoverable only by exploration with the finger. A second incision may be necessary to secure dependent drainage (*see* Fig. 142).

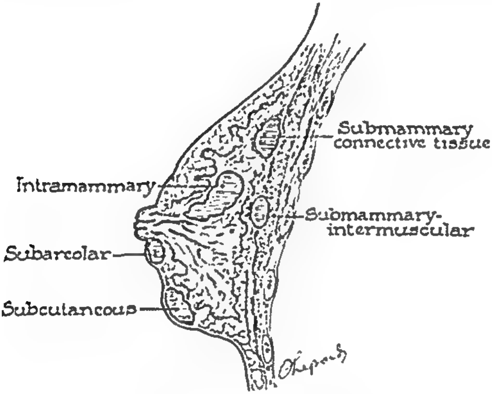

FIG. 141.—Locations of mammary abscesses.

Acute non-lactation mastitis is much less common and is treated similarly. When either sort of abscess is incised, but especially that not connected with lactation, it is wise to take a biopsy specimen of the abscess wall. If a lump remains after resolution of the acute inflammatory process, it must be removed for microscopic examination.

Chronic, nonspecific inflammatory lesions are uncommon but constitute a vexing problem. The inflammation may involve the areola or any quadrant of the breast. There is moderate redness, tenderness and edema. Usually there is a draining sinus which closes intermittently. Patients have often had previous surgical drainage with only temporary relief. These recurrent lesions are best treated by *excision* combined with the antibiotic and supportive adjuvant therapy.

Other chronic inflammations of the breasts are less common. They are duct ectasia, plasma cell mastitis, tuberculosis and syphilis in decreasing order of fre-

quency in our experience. *Only 9 cases of actinomycosis* have been recorded in the world literature. Ectasia of lacteal ducts presents clinically as single or multiple elongated, slightly tortuous, usually *firm serpentine* masses radiating from the areola. A thick green, *occasionally* purulent discharge may exude from the affected duct or ducts. If these lesions do not respond to conservative measures, excision may be necessary.

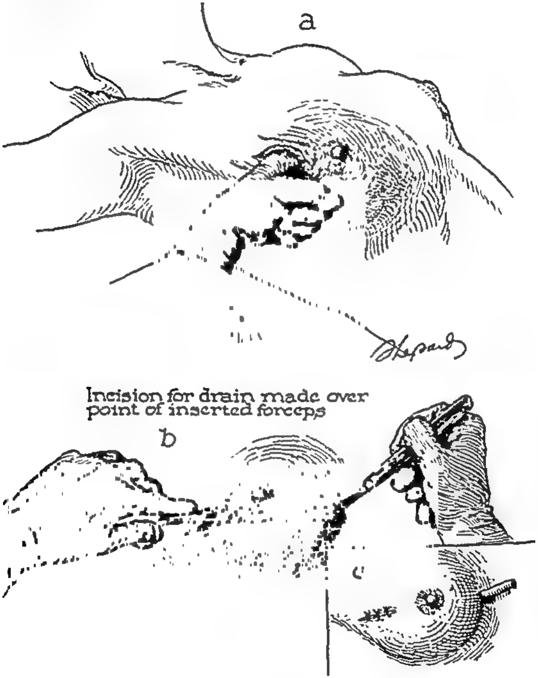

Fig. 142.—A method of incising and draining a mammary abscess.

Plasma cell mastitis is a rare inflammatory lesion associated with tenderness, edema, induration and fixation to the skin at the site of involvement. Axillary adenopathy may be present. The condition tends to resolve slowly and is differentiated from carcinoma only on microscopic study which reveals inflammatory cells associated with many plasma cells. Excision is curative. These two inflammations are of greatest importance because they can easily be mistaken clinically for carcinoma.

Tuberculosis of the breast resembles chronic nonspecific inflammatory lesions. One or more draining sinuses may be present. The disease involves all or a portion of the breast, and may clinically resemble diffuse carcinoma, or inflammatory carcinoma with axillary node involvement. In one of our cases the breast was fixed to the skin and chest wall. Axillary, supraclavicular and occipital nodes were enlarged and hard, and the skin over the breast was inflamed. Inflammatory carcinoma was the working diagnosis until biopsy revealed tuberculosis. The patient recovered on the medical management used for pulmonary tuberculosis.

CHRONIC CYSTIC MASTITIS

(Lumpiness and Lumps)

Chronic cystic mastitis is a lamentably inaccurate but familiar and conveniently inclusive term for breast disorders of functional origin. These dysplasias are characterized histologically by myoepithelial hyperplasia of varying degrees and clinically by sometimes tender fine granularity, nodularity, or areas of diffuse thickening. Mastodynia, mazoplasia, adenofibrosis, adenosis and Schimmelbusch's disease (partially equivalent term for chronic cystic mastitis) are manifested by these findings. The nodules are likely to be of about the same size, consistency and to feel about equally distinct or indistinct from adjacent breast tissue.

Although palpable changes of these sorts may occur in any or all parts of one or of both breasts they are most commonly felt in the upper outer quadrants, somewhat less commonly in the lower hemispheres where the breasts tend to fold over on themselves.

Associated tenderness or pain is frequently unilateral, even when the lesions are found in both breasts. Sensitivity and lumpiness are likely to worsen immediately before, and subside coincidentally with the onset of the menstrual periods, but either or both may persist longer. Large nodularity in the breasts of younger women is subject to the greatest periodic variations, while the fine, firm, shotty granularity in the pendulous breasts of older women is likely to be more persistent. The treatment of tender lumpiness consists of reassurance, firm support for the breasts and maintenance of the patient on a diet high in protein and the B vitamins.

Surgical biopsy, however, is required to distinguish some members of the chronic cystic mastitis complex from cancer. These are solitary cysts, aggregates of small cysts and foci of adenosis which are palpable as true lumps rather than lumpiness.

THE HISTORY AND PHYSICAL EXAMINATION

More than 90 per cent of breast cancers come to clinical notice because patients seek advice and treatment for complaints referable to the breasts, although a few cancers are found during examinations made for other reasons. The principal complaint is of lump. Most lumps are found quite accidentally but occasionally tenderness or pain causes patients to find them. Almost one-fourth of patients with carcinoma describe some sort of associated pain or tenderness.

The description of the duration of the complaints is often of suggestive value, since a cancer long standing without much evidence of growth or extension is likely to be only moderately malignant. A tender lump which appears all of a sudden in

the breast of a patient who is having slight menstrual irregularity is likely to be a solitary cyst. Th ecomplaints of nipple discharge or nipple eczema seldom lead to a diagnosis of cancer unless a lump is palpable at the time of the first examination. All other compaints are of importance only because they provide opportunities to find lumps during the complete examination of both breasts.

Antecedent Factors.—Knowledge of the age, race, parity, and personal and family data of breast cancer patients is of little practical use to the physician confronted by one patient with a breast lump. Some familiarity with age incidences and antecedent factors is necessary, however, in considering and discussing the possibilities. Most patients who consult physicians for symptoms referable to the breasts are less than 50 years of age. However, at least half the cases of cancer occur in the smaller group of older patients. Cancer is at least seven times as common as benign lumps in the breasts of women over 50, whereas before 40 the reverse obtains. During the years between 40 and 50, corresponding roughly to the menopause, benign and malignant lesions occur with about equal frequency. A majority of cases of breast cancer are found in the fifth, sixth and seventh decades, with onset before 30 a rarity, but the disease is seen often enough during the second and third decades of life to make it unsafe to say that any patient is too young to have cancer of the breast.

Previous breast history is of slight importance in the diagnosis of carcinoma, unless the patient describes the previous removal of a breast for cancer. (The disease is at least twice as common in the remaining breasts of cancer patients as in the population at large.) Few patients with breast cancer give a history of previous benign disease, although three out of four breasts removed for cancer show benign pathologic changes adjacent to the malignant ones.

There is some reason to believe that women who have had clinically evident cystic disease (micro-cysts included) are more likely than others to have cancer later, although it is not apparent that grossly evident cysts are much more frequent in carcinomatous breasts than in normal ones. Cancer does not necessarily occur in the same breast earlier involved with cystic disease. More women with clinically evident benign disease give a family history of breast cancer than do those whose breasts are normal, and family histories of breast cancer are even more common among patients who have that disease. Patients who have had previous operations upon their breasts or whose close female relatives have had breast cancer are likely to be diligent in the examinations of their breasts and somewhat more fearful of the possibilities of cancer in themselves. Practical considerations are that clinicians periodically repeat examinations on the breasts of such patients and remain keenly aware of the difficulty of finding small cancers in breasts which are generally lumpy.

The Examination.—The patient is first examined sitting in a good light and stripped to the waist. The examiner is not seeking signs pathognomonic of cancer, but visual evidences of lumps and of the most elusive earliest infiltration of cancer into Cooper's ligaments. This is faint dimpling or flattening to be seen on movement only, perhaps only in one particular position. Small abnormalities of contours of breasts, areolæ and nipples can be made out by altering the positions of patient or light source.

While the patient slowly raises and lowers her arms the examiner looks for deviations in the contours of the breasts, in the rhythm of nipple elevation, in the comparative level to which the nipples rise, and in the symmetry of their angles of pointing. Examination of the axillæ and of the supraclavicular spaces can best

be carried out while the patient is sitting. Palpation is most correctly done while the patient is reclining, and it is well to remember that inspection can be combined profitably with palpation. As the breasts are palpated they can be manipulated gently in the attempt to bring out the slight flattening or dimpling which is the first detectable sign of infiltrative growth. Gentle compression of the skin towards the lump may show skin adherence not otherwise demonstrable, and it may also make evident a small area of minimal skin edema. Much can be learned by careful inspection which might be missed if the examiner proceeded directly to palpation of the lump described by his patient.

The reclining patient is rolled slightly to the opposite side so that the breast under scrutiny is spread as evenly as possibly on the chest wall. It is easiest to examine the upper outer quadrant with the arm at the side, the others with the patient's hand behind the head. *Keep in mind that breast tissue may be found as* high as the clavicles, as far laterally as the anterior margins of the latissimus dorsi muscles, medially to mid-sternum, and as low as the rectus fascia. The axillary extension often reaches around the free border of the pectoralis major and into the axilla.

Palpation of the flattened out breast is most rewarding when done with the palmar surfaces of the adducted fingers, but small lumps beneath the nipple and areola may be difficult to feel in this manner. They are most likely to be detected when the nipple is rolled between the fingers with the patient sitting and the breast in the pendant position.

The nipple is squeezed gently between the fingers and noting the number and location of ducts from which discharge may be expressed, if any, as gentle pressure is applied circumferentially at increasing distances from the nipple. Long-continued low-grade inflammatory disease of the terminal ducts results in firm cordlike mass which can be felt centrally and which restricts the elasticity of the nipple. Prolongations of papillary growths into the terminal ducts may occasionally make a similar thickening, but papillomas are seldom large enough to feel when they originate in ducts within the nipple. Collections of fluid in dilated terminal ducts produces soft swellings which disappear as the fluid is pressed out of the involved duct or ducts.

Physical Signs of Cancer of the Breast.—Cancers generally present themselves as lumps which may be clinically indistinguishable from any other lumps in the breasts. About half of them are distinguished by palpable axillary nodes at the time they come to clinical notice. Some of these are still innocent of other stigmata of cancer but in a majority of instances the invasive activity of the cancer has produced additional signs suggestive of the nature of the disease by the time axillary spread has become evident. On the other hand it should be remembered that axillary nodes containing metastatic cancer can sometimes be felt before a breast tumor can be found (occult breast cancer), and that more rarely collapse of a vertebra from metastases may precede any symptoms referable to the breasts. Until infiltration and metastases have become apparent cancerous lumps cannot be distinguished from benign ones except by microscopy. Benign disease often enough acquires the signs of cancer so that mastectomy on the basis of clinical diagnosis alone is likely to lead to serious error.

The earliest sign of cancer additional to lump is faint dimpling noticed only upon movement of the breast (as distinct from a pucker present in all positions). Alteration in the axis of nipple pointing, in the contours of the breast, and the

slightest edema of areola or breast skin are indications of more advanced growth. By the time skin edema is easy to see or fixation to skin or to pectoral fascia can be demonstrated, multiple axillary nodes are usually palpable. Thus breast cancer which is readily recognizable as such is almost invariably advanced disease and likely to be incurable. It is obvious that surgery cannot be curative for cancer already spread beyond the confines of the breast and the axilla of the same side. It should be recognized by all surgeons that even though remote dissemination cannot be proved it can in many cases be strongly enough suspected to indicate that palliation only is to be gained in that particular case. Poor selection of cases for the radical operation accomplishes little if any good for patients, discourages the surgeon, and worsens the reputation of the operation.

Clinical Staging (Classification).—Attempts at clinical staging have as their objective the sorting of operable cases into two groups for the purpose of deciding which deserve radical operation as attempted cure, which can derive only palliative benefit from surgery (or other sorts of treatment). Obviously, those who propose simple mastectomy as a definitive operation have no need to classify their cases, since no effort is to be made to remove all the surgically accessible cancer. No method can be more accurate than our ability to feel small axillary nodes or to predict the extent of the disease from its local manifestations. The percentage of clinical error in both these efforts is at least 50 per cent, and this degree of inaccuracy is reflected in the mortality rate.

It is an obvious impossibility for the clinician who encounters suspicious breast lumps with average frequency to remember anything but the simplest grouping, hence he must have available an understandable description of breast cancer believed to be best treated by extensive operation with hope of long survival, and of that for which any surgery used should be palliative in intent. The following considerations regarding curability have served us well. In Clinical Class I are grouped those whose disease is thought to be limited to the breast alone, and those in whom it is believed one breast and the axillary nodes of the same side only are involved (Clinical Class II). Radical mastectomy is indicated for patients in these clinical Groups I and II. In the second group are those patients whose cancers can either be shown or presumed to have spread beyond the bounds of removal by the classical radical operation (Clinical Group III). The demonstration of cancer in the supraclavicular nodes, the opposite breast or axilla, the lungs, pleura, liver, bones, etc., is obvious evidence of surgical incurability as has been shown by numerous studies of post-mastectomy mortality.

Evidences of the presence of these remote metastases are found by careful examination of the opposite breast and axilla, the supraclavicular spaces, the liver region and pelvis. X-ray films of the chest show soft part detail, and those made with the Bucky diaphragm provide views of the thoracic vertebræ, the heads of the ribs, clavicles and upper ends of the humeri. The sites of any recently acquired bone pain or headache should also be filmed in the search for bony metastases.

Additionally, Haagensen, to whom we are indebted for many contributions on the surgery of breast cancer, showed by retrospective analysis of failures of the radical operation that there were local signs indicative of remote spread which could and should be used as contraindications to curability by the radical operation. He has stated these as follows: "Carcinoma of the breast in women of all age groups, who are in good enough general condition to withstand operation, should be treated by the Halsted radical mastectomy except as follows:

1. When extensive edema of the skin over the breast (more than one-third of the skin area) is present.
2. When satellite nodules are present in the skin over the breast.
3. When the carcinoma is of the inflammatory type.
4. When any two, or more, of the following grave signs of locally advanced cancer are present:
   a. Ulceration of the skin.
   b. Edema of the skin of limited extent (less than one-third of the skin over the breast).
   c. Solid fixation of the tumor to the chest wall.
   d. Axillary nodes measuring 2.5 cm. or more, in transverse diameter.
   e. Fixation of axillary lymph nodes to the skin or the deep structures of the axilla (includes ulceration).
5. When there is edema of the arm."

Haagensen originally included carcinoma developing during pregnancy or lactation as categorically incurable. Although the prognosis is extremely poor except for those patients who have no involved axillary nodes, current surgical opinion is that radical mastectomy is the operation of choice unless other evidence of incurability is found.

Radical operations undertaken in spite of the presence of these evidences of incurability do not increase the percentage of five-year survivals but do worsen the statistics of the operation. For the past five years Haagensen has used biopsy of the internal mammary and highest axillary nodes as a criterion of curability. He performs these biopsies concurrently with that of the primary tumor, and in those cases in which the likelihood seems greatest of finding metastases in these locations. Factors indicating this probability are axillary metastases, primary tumors of large size, and situation of the tumor in the central or medial half of the breast. If internal mammary or apical node carcinoma is found in paraffin sections, radiation therapy alone is used, otherwise delayed radical mastectomy is employed.

THE "EARLY" DIAGNOSIS OF BREAST CANCER

Survival of patients after operations for breast cancer depends upon many factors, of which the most important is the biologic variability of the disease (tumor-host relationship). Since the detection of lump is not necessarily coincidental with an early stage in the natural history of the carcinoma it is apparent that the time of operation cannot be the critical factor in survival nor can early diagnosis result in universal control of the disease. However, efforts to make the diagnosis and institute adquate surgical treatment as early as possible should be made since such efforts certainly result in salvage of some individuals who might be lost by erroneous clinical diagnosis, temporization and incomplete surgery. Thus every true lump, however small, must be removed for biopsy as soon as discovered. Lumps in thin flat breasts, or placed close to the surfaces of larger breasts, can be identified as such when only a few millimeters in diameter. However, most carcinomas are larger than this when found by clinicians and the average diameter of cancers removed in any one hospital is likely to be greater than two centimeters. It seems probable that a majority of carcinomas in excess of this size have metastasized at least to the axilla even though in more than one-third of instances these nodes may

not be palpable. Thus today's "early" diagnosis can be considered as such only when the tumors are bulky, slowly growing and slowly metastasizing. This situation could be improved by better education of patients and physicians.

BIOPSY

Biopsy for microscopic examination is the part of the diagnostic procedure which permits accurate identification of the nature of lumps. Error in this procedure is least probable when the tumor is removed by excision and a portion selected for microscopic examination after inspection of the entire mass and its cut surfaces.

If the tumor proves to be benign, the biopsy is a completed local excision. If a definite diagnosis of cancer cannot be made by examination of frozen sections, definitive treatment can be deferred until paraffin sections can be prepared. Delayed operations are not compromised by the healing biopsy wound.

The excision should include some adjacent normal tissue. If skin adherence is present, an ellipse of skin should be taken with the tumor. The incisions for surgical biopsy should be made with consideration for the cosmetic effect and for the need to avoid the lines of incision of possible subsequent radical mastectomy. They may be circum-areolar, curvilinear, transverse or radial. Incisions in the inframammary fold are satisfactory for the excision of tumors of the lowest part of the breast, but reflection of the breast upwards with a transmammary approach to lumps in other locations seems both poor surgical and cancer procedure.

Aspiration of tumor cells through a large needle with immediate examination of the stained smear is a simple but not too satisfactory method of biopsy for record purposes in cases of incurable cancer, when treatment will be nonsurgical. However, a better specimen for histologic examination can be secured by cutting a core of tissue with the Vim-Silverman needle. Incision directly into, and excision of a small piece from very large or incurable or inoperable tumors avoids unnecessarily extensive procedures.

Novocain skin infiltration may be used for needle biopsies, but general anesthesia is preferred in other biopsy procedures. Patients should be told that since decision concerning the desirable extent of operation cannot be made until after the microscopic examination their permission for mastectomy must be secured before they are put to sleep for the biopsy.

Because of the danger of implantation of cancer cells into the wound, the gross pathologic examination of the specimen is made by the surgeon after he has left *the operating table and the wound is closed by assistants.* If mastectomy is indicated by the microscopic findings, cancer asepsis is better served if the entire operative field is gently recleansed and a complete change of drapes, gowns, gloves and instruments is made.

The Aspiration of Cysts.—Lumps which appear suddenly, have an elastic feel, transilluminate or can be shown by radiography to be cysts, may be aspirated. If they flatten completely after the withdrawal of nonbloody fluid, further treatment may be deferred pending a second examination four weeks later. Subsequently formed cysts may be aspirated repeatedly under the same conditions. Generally it is better to resect the first occurrence of cyst and to aspirate subsequent ones. Surgical biopsy is indicated when bloody fluid is aspirated, a lump remains after aspiration, or recurrence appears at the same site.

Biopsy for Nipple Discharge Without Lump (Babcock Technique).—Under general anesthesia a blunted 22-gauge 3-inch needle is worked down the involved duct, being allowed to take its own direction towards the periphery. A circumareolar incision, centered on the needle direction, allows exposure and resection of the involved duct through a radial division of the breast substance. Serous or bloody discharges from the nipple are usually due to intraductal papilloma or to papillomatosis. Both these benign conditions occasionally make a perceptible lump which disappears as it is pressed, with the fluid being squeezed out the duct. Carcinomas growing in ducts seldom cause discharges from the nipple without manifesting themselves as lumps. Discharges other than serous or blood are of negligible importance in the diagnosis of carcinoma. If lump and discharge both are present, proceed as for lump.

Biopsy in Paget's Disease.—Eczema of the nipple which fails to heal in one month should be subjected to surgical biopsy of the involved skin. Ulcer of the nipple without lump requires excision and careful study of the sections through the terminal ducts. A palpable lump, when present, must be excised with nipple and areola. The eczematous or ulcerative changes are the result of carcinoma growing in the lactiferous ducts. Axillary nodes are palpable in at least half the cases at the time of first examination.

Biopsies During Pregnancy.—Lumps coming to notice during pregnancy are biopsied and the indicated treatment carried out until the fifth month of gestation. Later than this we have generally delayed the biopsy until after delivery by section at the time of viability. In most of our young patients whose tumors appeared almost certainly to be fibroadenomas we have delayed the biopsies until delivery at term.

PATHOLOGY

Lumps removed at biopsy have gross characteristics which allow experienced surgeons and pathologists to recognize about nine out of ten carcinomas by their feel and appearance. However it is probable that experts who claim never to have made a mistake in the gross pathologic recognition of the nature of breast lumps have forgotten at least one such error. Thus final diagnosis and mastectomy are never justified without the examination of microscopic sections. Nonetheless, it seems appropriate here to include a few paragraphs on the gross pathology of breast lumps.

Carcinoma.—Most carcinomas are hard, indistinctly outlined masses. The knife grates in them, and when it is stroked across the cut surfaces it makes a scratching noise. The cut surfaces are concave, dirty gray-white, streaked with pale yellow. It is usually easy to see that the tumor is growing into adjacent breast substance, although some of the softer ones compress surrounding breast tissue by their rapid growth and thus may give the appearance of encapsulation. About one-eighth of them are softer than the common "scirrhous," these are the more cellular, predominantly papillary, colloid or medullary types. These types are thought to have a slightly better prognosis than the histologically poorly differentiated types, but neither the gross nor the microscopic appearances of carcinomas are of moment in selection of the type of operation to be performed, which must be done strictly on clinical grounds.

It is important, however, that the surgeon recognize the disease known as inflammatory carcinoma, for no good comes of operating on this disease. A limited

area of redness without heat over a small breast tumor should not be considered inflammatory cancer, but diffuse redness and heat, associated with a larger tumor and palpable axillary nodes usually proves to be rapidly fatal regardless of treatment. Occasionally a febrile course has been noted in connection with this disease.

Benign Tumors of the Breasts.—The common indigenous tumors of the breasts are fibroadenoma and papilloma. Fibroadenomas sometimes occur as multiple tumors, less commonly as rapidly growing, large lobulated masses. When these *giant fibroadenomas* undergo great increases in the amount and cellularity of their stroma, with a microscopic appearance suggesting sarcoma, they are called *cystosarcoma phylloides*. Most of them behave as benign tumors and are cured by simple mastectomy. They occasionally ulcerate through the skin.

Fibroadenomas are encapsulated, often are extruded in front of the breasts, and slip around freely under the fingers. The surfaces made by cutting tend to bulge, are pearly white, and show slits and whorls corresponding to the amount of intraductal and peri-canalicular growth present.

Cysts and Fibrocystic Disease.—These lesions have been discussed in the section on physiology. Inflammation around cysts, as around any other benign breast lesion, can produce the dimpling and other contour changes considered characteristic of cancer.

Fat Necrosis.—This unusual lesion, most often due to trauma, whether this is remembered or not, somewhat resembles carcinoma in clinical, gross and, to a lesser extent, in microscopic character.

Lipomas.—Lipomas are the only common non-indigenous tumors of the breasts and have the clinical characteristics of like tumors occurring elsewhere.

RADICAL MASTECTOMY

The operation for the removal of breast cancer with the entire breast and all its possible axillary metastases from the chest wall in one piece was formalized over 60 years ago by Halsted and by Willy Meyer. When performed with attention to the logical details first enunciated by them, radical mastectomy is a tedious and somewhat difficult procedure, the use of which is justified only when there is strong probability that all the cancer is within the field of excision. The operation has commonly been misused for obviously disseminated and hopeless cancer and unmercifully modified by restriction of its extent when used for "early" carcinoma.

Students and clinicans have had reason for some confusion concerning the worth of the operation because of conflicting opinions freely expressed, often without critical study of long-term results. Surgeons who for one reason or another favor simple mastectomy have considered the radical operation too extensive. These surgeons have been greatly receptive to the idea of simple mastectomy followed by deep *x*-ray therapy as definitive treatment for cancers of any stage. Lately others, coming to believe the internal mammary nodes regional rather than remote, have considered the standard operation to be not extensive enough. Statisticians have challenged the validity of the five-year survival as a measure of the efficacy of the operation. Yet the consensus of the literature is that radical mastectomy is the operation of choice for cancer believed to be limited to the breast and axilla (Clinical Groups I and II).

The operation subsequently to be described is the one taught to the residents of the Breast Service at the Cook County Hospital. It includes features of the opera-

tion performed by many different surgeons, but is based on the principles enunciated by Halsted and Meyer. These are removal *en masse* of the skin over and surrounding the tumor, the entire breast, both pectoral muscles, the axillary fascia, fat, lymphatics and nodes. It is neither as detailed and minutely meticulous a procedure as described by some, nor a hasty, roughly performed blunt dissection such as is occasionally seen. The description of this operation is offered without any attempt to answer criticisms of details or to reiterate anatomic justification for each step. We believe that by its use we have reduced the incidence of local persistence and recurrence, caused less arm disability and cured some patients with axillary metastases. We believe this method permits excellent exposure with minimum handling of the breast, that it can be accomplished within three hours with slight morbidity and less than 0.5 per cent mortality directly attributable to the operation.

Contraindications.—An operation of this magnitude is not justifiable when palliation only can be gained (*see* discussion of Criteria of Incurability). It should not be attempted in the cases of patients who have concurrent diseases likely to be fatal in a matter of months nor which contraindicate general anesthesia. Advanced age is not of itself a contraindication to radical mastectomy. Since a majority of patients coming to surgery for breast cancer are in the sixth and seventh decades many are found to suffer also from the degenerative diseases of middle life and senescence. Operation may occasionally have to be deferred temporarily because of cardiac decompensation, recent coronary heart disease, or poorly controlled diabetes. These conditions, as well as secondary anemia, chronic renal or hepatic insufficiency, and the susceptibility of older patients to pulmonary infections increase liability to postoperative complications. Careful preoperative clinical and laboratory evaluation, fluid, electrolyte, and blood replacement plus skillful anesthetization allow avoidance of most operative and postoperative complications, and keep the mortality rate at less than 1 per cent.

Anesthesia for radical mastectomy should be by inhalation, utilizing cyclopropane, ethylene, nitrous oxide or ether, preferably one of the latter two. If electrocoagulation is used for bleeders on the breast side, or an electric skin grafting machine is to be employed, the anesthesia during these periods must be nitrous oxide-oxygen. Since the operation requires no relaxation, light anesthesia is desirable. The use of a barbiturate the night before and two hours before operation allows a good night's sleep and allays pre-anesthetic apprehension. Morphine or demerol with scopolamine, given one hour preoperatively, aid induction and render the patient carefree and forgetful. Pentothal may be used for induction but is not advised for continous administration in anesthetic doess during a long operation. The use of an endotracheal tube lightens the work of respiration and allows lighter anesthesia. Closed circuit endotracheal anesthesia is a must for *extended* radical mastectomy.

The operative field is prepared by shaving of the axilla and the breast, then gentle washing for at least seven minutes with solutions of G-11 soap of the supraclavicular fossa, the involved half of the chest, extending over almost to the opposite areola, the upper abdomen, the axilla and the arm down to the elbow. The thigh of the same side is also prepared in the same manner as a donor site. All preparatory measures are carried out as gently as possible so as not to massage the tumor with untoward results. The arm is placed on a narrow rest at right angles to the trunk with the patient well over to that side of the table.

When radical mastectomy follows biopsy, gentle rewashing of the operative

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When radical mastectomy follows biopsy, gentle rewashing of the operative

field, change of all linens, gowns, gloves and instruments is required. These procedures are facilitated if the surgical nurses have had special instruction in the preparation and draping of patients for this operation.

The Incision.—There is no standard incision for radical mastectomy. An incision adequate for removal of all possibly involved skin must encircle the tumor at least two inches away from its palpable margins. In many cases this will include most of the skin of the protuberant breast. Upper and lower extensions from this encircling incision must allow access by retraction of the flaps to the pectoralis major tendon, to the apex of the axilla and to the other limits of resection of breast and muscle. The upper extension of the incision should not run across the axilla nor out onto the anterior surface of the arm because of the postoperative interference with arm motion. If the upper part of the incision runs downward from about the midpoint of the clavicle, the scar will be covered by the brassiere strap.

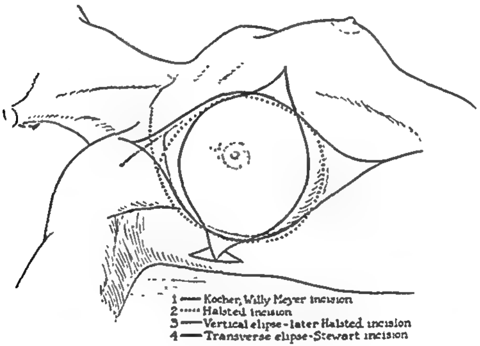

FIG. 143.—Various incisions for resection of the breast.

Surgeons differ in their preference for incisions, and many use the incision first described by Rodman (*see* Fig. 144). The transverse incision of Stewart does not give good enough exposure of the axilla to be satisfactory for radical mastectomy, although it serves well for simple mastectomy, especially in obese subjects (Fig. 143).

The lines of the projected incision may be marked on the skin with the dull side of the knife. The lateral component is made by cutting just barely through the skin so that the flap can be raised by a dissection at the level of the superficial fascia. This dissection, making the flap but little thicker than a full-thickness graft, is carried laterally while the breast is retracted medially by means of the skin clips which hold a towel to the margin of the skin over the tumor. Keeping the dissection at this level insures that no breast tissue will be left on the flap and that beeding from both breast and skin will be minimal. The level is not deepened until the anterior margin of the latissimus muscle can be found. Superiorly the flap extends to the clavicle, inferiorly to the rectus sheath (Fig. 145).

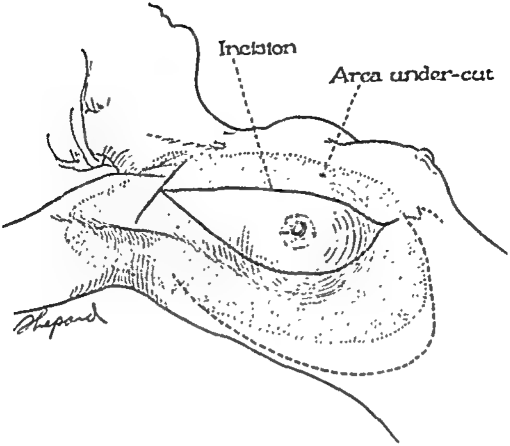

FIG. 144.—Scheme of Rodman's incision.

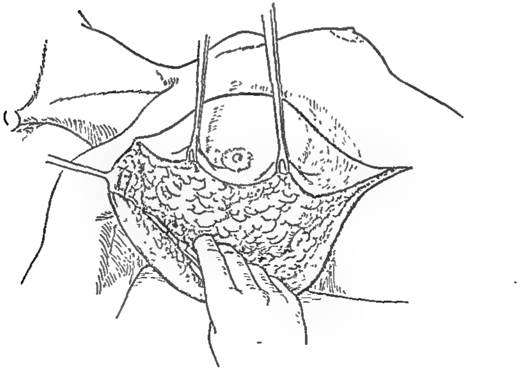

FIG. 145.—The lateral incision has been made and the flap is being reflected by sharp dissection.

Retraction of the margins of the flap is accomplished by sharp rake retractors or by fine skin hooks. Dissection proceeds downward to the lateral and inferior margin of the pectoralis major tendon and upward to the tendinous portion of the latissimus dorsi leaving the freeing of the flap from the axillary fat for the last. This portion must be thin, developed so as to show apocrine sweat glands in the axillary skin. Bleeders on the flap are grasped with fine pointed Kelly forceps and ligated with 0000 silk. Those on the breast side may be tied more crudely, or coagulated electrically. Hemostasis accomplished, the gutter is packed with a hot lap sponge and the breast allowed to fall laterally.

The medial incision is now made and the medial flap developed in the same manner as the lateral one, carrying the dissection to the midline of the sternum and down onto the aponeurosis over the fibers of origin of the rectus muscle. The extent of the flaps is illustrated in Figure 146.

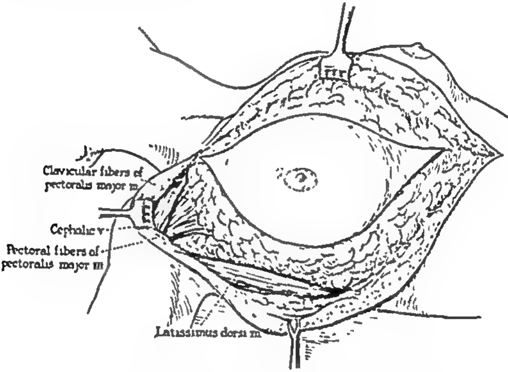

FIG. 146.—The lateral flap has been reflected exposing the latissimus dorsi muscle and the lateral limits of the dissection.

For the last time the breast is raised medialwards, the pectoralis tendon exposed, and the pectoral fibers divided close to their insertion, leaving clavicular fibers intact (Fig. 147). The division between pectoral and clavicular fibers slightly caudad to the cephalic vein is carried medially toward the sternoclavicular junction, clamping, dividing, and ligating pectoral vessels and the anterior thoracic nerves going to the pectoralis major. The medial flap is retracted and the fat and fascia are incised down to the midline of the sternum from the sternoclavicular junction to the rectus origins. The operator retracts the mass caudally and laterally as he cuts the fibers of origin of the pectoralis major free from the sternum, rib cartilages, ribs and intercostal muscles (Fig. 148). As the fascicles of origin are cut, the first assistant clamps the perforating branches of the internal mammary artery with their veins as they are skeletonized and put on the stretch over the superior margins of the ribs. This dissection separates the pectoralis major origins and breast from

the chest wall well down onto the rectus sheath, farther than is shown in the figure. As it proceeds laterally, the less sturdy origins of the pectoralis minor come into view and must be cut from the surfaces of the third, fourth and fifth ribs. Small perforating vessels must be clamped before cutting while this division is taking place and the interdigitating fibers of the serratus anterior must be left in their place on the chest wall. Thus freed, the entire mass to be removed slides laterally, putting the deep fascia anterior to the axillary contents on the stretch. This

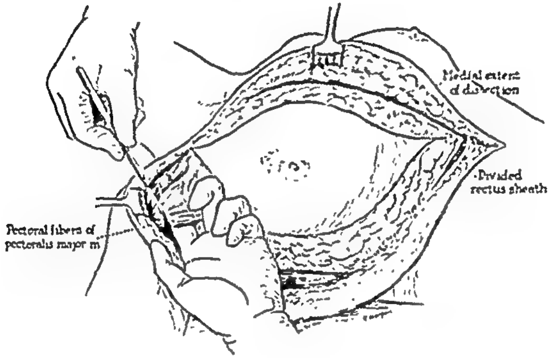

Fig. 147.—Pectoral fibers are being cut close to their insertion on the lip of the intertubercular sulcus of the humerus.

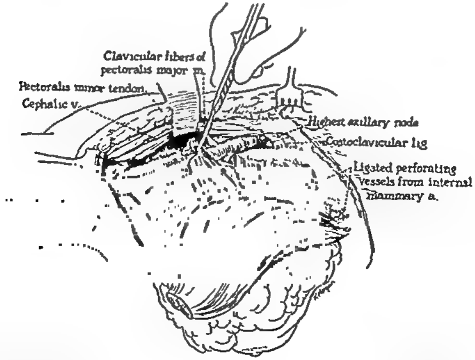

Fig. 148.—The tendon of the pectoralis minor muscle is being cut close to the coracoid process after the origin of this muscle and the major muscle have been incised from the ribs.

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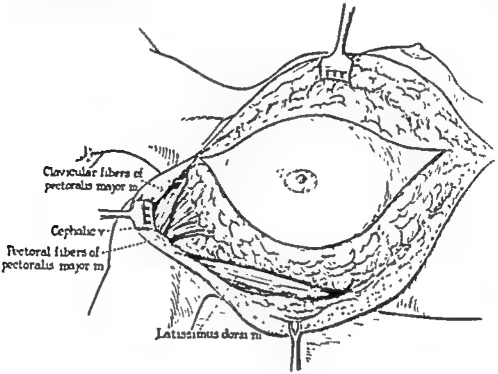

FIG. 146.—The lateral flap has been reflected exposing the latissimus dorsi muscle and the lateral limits of the dissection.

For the last time the breast is raised medialwards, the pectoralis tendon exposed, and the pectoral fibers divided close to their insertion, leaving clavicular fibers intact (Fig. 147). The division between pectoral and clavicular fibers slightly caudad to the cephalic vein is carried medially toward the sternoclavicular junction, clamping, dividing, and ligating pectoral vessels and the anterior thoracic nerves going to the pectoralis major. The medial flap is retracted and the fat and fascia are incised down to the midline of the sternum from the sternoclavicular junction to the rectus origins. The operator retracts the mass caudally and laterally as he cuts the fibers of origin of the pectoralis major free from the sternum, rib cartilages, ribs and intercostal muscles (Fig. 148). As the fascicles of origin are cut, the first assistant clamps the perforating branches of the internal mammary artery with their veins as they are skeletonized and put on the stretch over the superior margins of the ribs. This dissection separates the pectoralis major origins and breast from

the chest wall well down onto the rectus sheath, farther than is shown in the figure. As it proceeds laterally, the less sturdy origins of the pectoralis minor come into view and must be cut from the surfaces of the third, fourth and fifth ribs. Small perforating vessels must be clamped before cutting while this division is taking place and the interdigitating fibers of the serratus anterior must be left in their place on the chest wall. Thus freed, the entire mass to be removed slides laterally, putting the deep fascia anterior to the axillary contents on the stretch. This

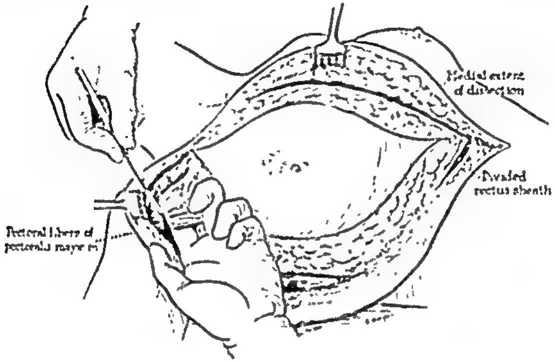

FIG. 147.—Pectoral fibers are being cut close to their insertion on the lip of the intertubercular sulcus of the humerus.

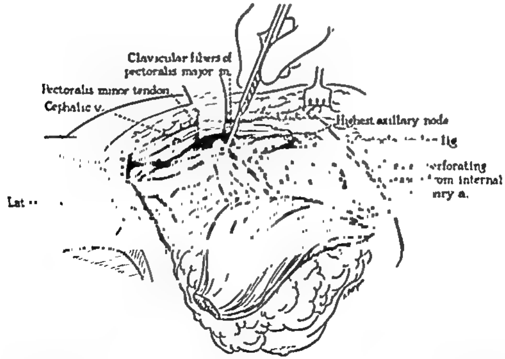

FIG. 148.—The tendon of the pectoralis minor muscle is being cut close to the coracoid process after the origin of this muscle and the major muscle have been incised from the ribs.

fascia (anterior axillary, deep pectoral) encloses the *pectoralis minor* tendon and stretches medially to the costoclavicular junction, laterally to the *coraco-brachialis* muscle.

The tendon of the pectoralis minor muscle is *identified beneath* the anterior axillary fascia, which is incised to expose it so that it *may be divided close* to its insertion on the coracoid process. A small *artery entering the muscle* on its medial aspect requires ligation. The division of the *deep axillary fascia* made to expose the *pectoralis minor* tendon is carried medially and laterally parallel to the upper *margin of the axillary* vein which can be seen beneath it (Fig. 149 shows this *division already* accomplished). Medially the dissection is carried up to a thickened portion of the fascia over the subclavius muscle, the costoclavicular ligament.

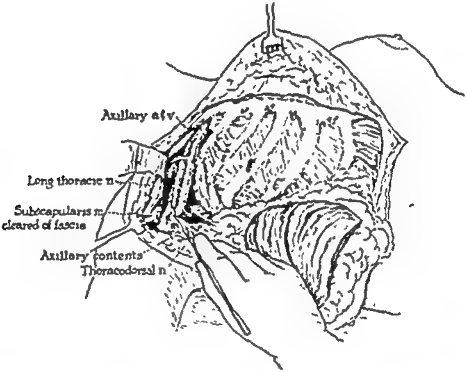

FIG. 149.—The axillary vein has been cleared by sharp dissection and the fascia of the subscapularis muscle is included with the specimen.

No retractors are necessary for the dissection of the axilla. The weight of the breast and muscles draws the axillary contents outwards and downwards as they are dissected off the adventitia of the vein and the lateral chest wall with the knife. A fresh blade should be used for this dissection. All tributaries entering the vein are clamped, divided, and ligated immediately as are a few small arteries crossing the vein to enter the pectoralis muscles. As the fascia is dissected backwards from the serratus fibers on the lateral chest wall the long thoracic nerve is seen lying beneath its surface. The fascia is separated from the nerve and this dissection continued backward to the angle between serratus and subscapularis muscles.

The removal of the axillary fat from the vein extends backwards to the subscapularis fascia where it meets the dissection just described. Laterally, the vein is cleared to the level of the tendon of the latissimus dorsi tendon, where the thoracodorsal nerve is encountered (Fig. 149). If palpable lymph nodes exist in this region, the nerve is sacrificed, otherwise it is dissected free of the axillary fat. The separation of the remaining axillary fat from the lateral chest wall, the anterior

surface of the subscapularis muscle, and the lower portion of the latissimus dorsi brings the dissection into the gutter ("the bloody gulch") between the chest wall and the latissimus dorsi where attention must be given to securing branches of the subscapular vessels and to avoiding damage to the long thoracic nerve. This completes the dissection and allows the breast to slide into the specimen basin held at the side. A small stab wound is made in the skin of the lateral flap in front of the anterior margin of the latissimus dorsi tendon and a Foley catheter is drawn out through this so that the bulb and tip will rest in the axillary space. The skin

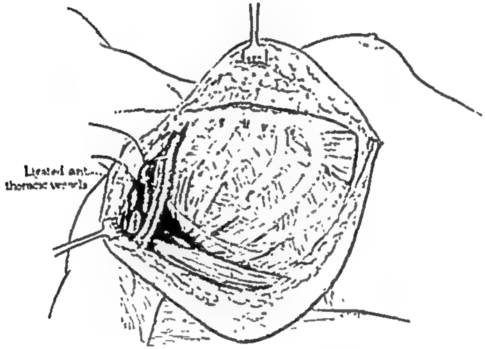

Fig. 150.—The completed radical dissection.

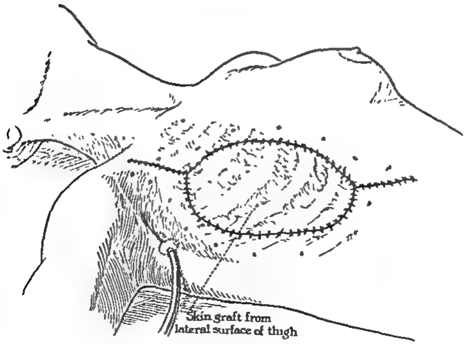

Fig 151.—The closure of the wound with skin graft and suction drainage of axilla through a Foley catheter.

nation of methods produces survival percentages equal to those gained by radical mastectomy alone.

Radiotherapy is the treatment of choice for soft part and bony metastatic lesions.

Radioactive Gold.—Radioactive gold (and nitrogen mustards) occasionally give dramatic relief for recurrent pleural effusions due to breast cancer metastases.

Endocrine Therapy.—Palliation of incurable breast cancer to a variable degree can be obtained by alteration of the hormonal environment. The simplest means of accomplishing such change is by the administration of estrogenic and androgenic hormones. Testosterone propionate is most commonly used for bony metastases in patients of all ages, and for soft part metastases in women whose menopause has occurred less than five years previously. Estrogens have been used mainly for attempted control of metastases in women more than five years past their menopause. Although the side effects of estrogens are less distressing than those of androgens, they are considerable. Nausea and vomiting, or uterine bleeding, occasionally require abandonment of the treatment. Voice changes, hirsutism, and increased libido are unwelcome side effects of the androgens. Testosterone propionate is given in doses of from 150 to 300 mgs. weekly, and estrogen, as stilbesterol, in doses of 15 mgs. daily.

The endocrine environment can also be altered by radiation suppression of ovarian function or by oöphorectomy. Neither of these procedures has proved of prophylactic value and their use should be reserved for incurable carcinoma. It is widely believed that oöphorectomy produces some palliation in approximately a third of those instances in which it is so used.

Adrenalectomy and hypophysectomy are used occasionally for palliation when patients survive after all other methods have failed. Occasionally rather remarkable regression of metastases has followed these procedures, particularly that of adrenalectomy combined with oöphorectomy (*see* Chapter 22).

QUESTIONNAIRE

1. Describe briefly the development of the breast.
2. What is the "axillary tail of Spence"?
3. What are acini?
4. What is the name of the connective tissue surrounding the acini, and that surrounding the ducts?
5. What are the ligaments of Cooper?
6. What is their clinical significance?
7. Give the location and distribution of the principal axillary lymph nodes.
8. What is the blood supply of the breast?
9. What is the nerve supply of the breast?
10. Discuss the role of hormones in breast diseases.
11. In which age-group is cancer of the breast most prevalent?
12. [illegible] breast tumors.
13. [illegible] e diagnosis of breast tumors?
14. [illegible] lischarge from the nipple?
15. How much importance would you attach to a serous discharge associated with a lump in the breast?
16. What is the significance of a greenish discharge?
17. What important points should be observed when inspecting the breast?
18. What does irregularity of breast outline signify?
19. Describe the technique of palpating the breasts.
20. What should you notice when palpating the breast?
21. Discuss the value of biopsy.

22. Classify breast abscesses as to location.
23. How would you treat a breast abscess?
24. Discuss the signs, symptoms and treatment of acute non-lactating mastitis.
25. What are some terms applied to chronic cystic mastitis?
26. Discuss the etiology of this disease.
27. Give the signs and symptoms of cancer of the breast.
28. Define "peau de orange."
29. What factors determine the prognosis of cancer of the breast?
30. Discuss the signs and symptoms of Paget's disease.
31. Discuss the clinical staging of breast cancer.
32. List the criteria of incurability.
33. Should radical mastectomy be employed when these signs are present?
34. Describe the Rodman incision.
35. Describe the technique for the radical resection of the breast.
36. Discuss incisions commonly used for radical mastectomy.
37. Describe use of suction drainage after radical mastectomy.
38. Discuss *x*-ray treatment subsequent to radical mastectomy.
39. List methods of altering the hormonal environment of cancer of the breast.

CHAPTER 22

ADRENALECTOMY

By Louis River and Joseph Silverstein

The adrenal glands are made up of a medulla composed of chromaffin tissue, which secretes adrenalin, and a cortex related to the sex organs, which secretes steroids. Tumors of the adrenal gland may take their origin from the cortex or the medulla. These tumors may be functioning or nonfunctioning. The latter tumors, particularly the cortical adenomas, are generally small and found at autopsy. Tumors that may give rise to Cushing's syndrome in adults develop in the cortex. Feminizing malignant tumors of the cortex may develop in the male. The adrenogenital syndrome (puberty precox) may develop in children as the result of benign or malignant adenomas or as the result of hyperplasia of the cortex. The tumors of the adrenal medulla may be neuroblastoma, benign and malignant, paragangliomas (pheochromocytomas), chromaffinomas and ganglioneuromas. The above tumors are indications for partial or subtotal adrenalectomy. In the absence of a tumor but in the presence of adrenal hyperfunction, a subtotal adrenalectomy may be performed.

It has been observed that certain breast carcinomas are affected by ovarian hormones, presumably by the estrogens. Oöphorectomy has been performed in patients with breast carcinoma who have demonstrable metastases and recurrences. On occasion this has resulted in definite objective evidence of improvement; many metastatic or recurrent nodules are reduced in size; bony metastases may actually show evidence of healing. These results, when they occur, do so more commonly in the pre-menopausal patient. Though it has not been our practice to do so, some surgeons prefer to do bilateral oöphorectomy prophylactically in premenopausal patients with breast carcinoma. Others use *x*-ray ablation of the ovaries. It has not been demonstrated satisfactorily that any given type of tumor will respond to such surgery consistently. It is believed that tumors and metastases with glandular configuration histologically may regress more readily with such therapy. *Unfortunately oöphorectomy has not served to cure the patient* with breast carcinoma. It has been found that the patient who does respond more favorably to oöphorectomy is the one more likely to respond to bilateral adrenalectomy.

Adrenalectomy is recommended in those patients in whom there are recurrences and evidence of metastases—and in whom other therapy has been used to a point of exhaustion, namely—local *x*-ray therapy and steroids. Where these modalities have been given fair therapeutic trials with no further evidence of tumor regression, or with progressive tumor spread, it is recommended that bilateral adrenalectomy be done. One investigator has found that the patient who has evidence of continued estrogen output, as measured in the urine, following oöphorectomy, is the patient to be more likely benefited by adrenalectomy. It is known that the adrenal cortex is a source of estrogenic hormone. It should be pointed out that the adrenalectomized patient has been converted to an Addisonian state, and as such is dependent

upon cortisone therapy for the remainder of her life. It should also be emphasized that this form of therapy is not curative but rather palliative in nature, its intent being to reduce the suffering of pain, initiate regression of metastatic sites and prolong life. Its specific value has yet to be proved.

Operations upon the adrenal glands can be performed through intercostal (transpleural, transdiaphragmatic), lumbar and abdominal incisions. The last-named approach affords four major advantages over operations by other routes: (1) Both glands may be examined or removed through a single incision and without changing the position of the patient. (2) Abdominal and pelvic exploration may be made a part of the operation, with search for aberrant adrenal tissue, and for tumors arising in that or in other organs derived from the urogenital ridge, bilateral oöphorectomy can be performed. (3) The adrenal veins may be ligated and divided without direct manipulation of the glands, and (4) novocain may be injected under direct vision into retroperitoneal spaces in celiac and perirenal areas. The latter maneuver provides complete relaxation with light inhalation anesthesia and avoids untoward stimulating effects of manipulations close to the diaphragm. The technique described is practicable in the cases of patients with a variety of body builds, even in those with narrow costal angles or with considerable obesity of the abdominal wall. We believe it to be the method of choice for exposure or removal of both adrenals.

ANATOMY

Since displacement of important structures in anterior relationship to the adrenals is an essential feature of access to the glands from in front, it is important briefly to review the surgical anatomy involved. The adrenals are enclosed in special compartments of the peri-renal fascia (Gerota's capsule) continuous with that surrounding the kidneys. They remain in close approximation to the kidneys as long as ptosis of the latter does not occur. The adrenals differ slightly in their relations and in their shape. The right is pyramidal or conical, capping the highest portion of the anteromedial border of the kidney. Its hilus is overlapped by the right margin of the inferior vena cava, which thus conceals the short, anterior, adrenal vein emptying into its posterior aspect. Usually this vein may be longer and run downward, even to empty into the right renal. Laterally to the vena cava the first portion of the duodenum overlies the antero-inferior surface of the adrenal; its anterolateral surface is in contact with the right lobe of the liver near the so-called bare area. Thus the right gland may have no contact with peritoneum.

Posteriorly and superiorly each adrenal lies upon the lumbocostal arch and near the corresponding crus of the diaphragm. Although the right gland is slightly more caudad with relation to the posterior abdominal wall, access to it from in front is more difficult than access to the left gland because of the former's position behind the liver and vena cava. The longer, crescent-shaped left adrenal lies more medially to the upper pole of the kidney. Its longer vein courses downward to empty into the left renal vein, and is exposed by division of the posterior peritoneum of the omental bursa which covers the gland anterosuperiorly, and by downward retraction of the tail of the pancreas and splenic vein. Not only is the left adrenal vein easier to expose than the right but it also less restricts mobilization of the gland. We have not found it possible to adequately explore the adrenal glands by palpation while the posterior peritoneum is till intact.

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The adrenal arteries are numerous and generally small. They are derived from

the aorta and the inferior phrenic and renal arteries and enter the glands at their borders. Their number, size and derivation are greatly variable, hence isolation and individual ligations are difficult and unnecessary.

OPERATIVE TECHNIQUE

Inhalation anesthesia is supplemented by infiltration of 0.5 per cent novocain *along costal margins, beneath diaphragmatic peritoneum* and around the celiac axis and the superior poles of the kidneys. An "inverted V" bilateral subcostal oblique incision two fingerbreadths below the costal margin is carried through all the structures of the abdominal wall. The divided rectus fibers are ligated in bundles and the falciform and round ligaments of the liver are clamped, cut and ligated. The gastrocolic omentum is divided just below the greater curvature of the stomach at the left, and vasa breva ligated and cut as necessary to allow retraction of the stomach upward and to the right, and of the splenic flexure and transverse colon downward and to the left. Occasionally this latter movement requires division of the lateral leaf of peritoneum and downward displacement of the spleen. The tail of the pancreas and the splenic vessels can now be seen overlying the lower portion of adrenal and the renal vessels; after incision of the posterior peritoneum transversely they are retracted downwards. If their downward dislocation is hindered by the left gastro-epiploic vessels, these may be divided.

The fingers are insinuated behind the kidney slightly lateral to the superior pole, bringing it forward and downward. A small lap sponge or narrow packing introduced into this space as the fingers are withdrawn maintains the more accessible position. A long Mixter forceps carrying a 24-inch length of Penrose tubing is passed superiorly to the renal vessels between and posteriorly to the superior pole of the kidney and the inferior pole of the adrenal to be caught and carried anteriorly at the junction of their superior poles. Gentle traction on this sling around the extension of Gerota's fascia between the two organs rotates the superior pole of the kidney forward and downward, bringing and holding the adrenal at least 2 inches anteriorly into the field. The adrenal vein, usually visible on the anterior surface of the gland, is divided between ligatures, after which three ligatures are carried between adrenal margins, and diaphragm, great vessels and renal vessels, respectively, enclosing the adrenal arteries. As these are tied the adrenal is cut free along its borders, requiring only the division of the fascial extension between the kidney and itself to come free. No vessels of any importance are encountered during this last division. The kidney falls back into place and after inspection of the field, usually dry, all other retraction is released.

To expose the right adrenal the omentum, transverse colon and duodenum are *retracted downward and medially* so as to carry the free right margin of the hepatoduodenal ligament to the left. The posterior peritoneum is identified where it is reflected off the bare area of the liver, it is incised vertically just to the right of the inferior vena cava and as far caudad as the renal vein. The right margin of the vena cava is retracted medially and the right adrenal vein sought behind it. The vein is cut between ligatures placed as close to the gland as possible. The Penrose tubing is then passed as on the left and the superior pole of the kidney, with the adrenal, dislocated upward into the operative field to facilitate the passing and tying of the three ligatures on the arteries. Lastly, the adrenal is cut free from the kidney completing the operation. Tension across the wound noted during the closure

may be lessened by placing sandbags under shoulders and buttocks. Interrupted wire sutures are used in the closure of the anterior rectus sheath.

The operation of bilateral adrenalectomy is no more difficult than are many upper abdominal procedures more frequently performed. However, avoidance of morbidity and mortality in the surgery of the adrenal glands depends not only upon good operative technique but particularly upon the accuracy of medical management before, during and after surgery. Details of this latter should be sought in papers on adrenalectomy for breast cancer palliation, pheochromocytoma, and Cushing's disease.

QUESTIONNAIRE

1. Where do tumors of the adrenal gland originate?
2. Which type of tumor may give rise to Cushing's syndrome?
3. Which tumors may require a partial or subtotal adrenalectomy?
4. Discuss the role of oöphorectomy in patients with breast cancer.
5. When is adrenalectomy indicated in breast cancer?
6. Discuss the rationale of this form of treatment.
7. Is it a curative or palliative form of treatment?
8. Give the various routes by which adrenalectomy may be done.
9. What are the advantages of the abdominal approach?
10. Give the surgical anatomy of the adrenal glands.
11. Describe the technique of unilateral and bilateral adrenalectomy.

CHAPTER 23

SURGERY OF THE HEART AND GREAT VESSELS

Introduction

BY JOHN L. KEELEY

CONSTRICTIVE PERICARDITIS

BECAUSE of the tremendous strides made in the field of cardiovascular surgery in recent years what is written about this surgical specialty today may not be tomorrow's approach to the same problem. This is particularly true of those intracardiac lesions which have been attacked in the past by indirect methods but which are now being approached by open cardiotomy during interruption of circulation, either under hypothermic conditions or by maintenance of cardio-respiratory function with extra-corporeal apparatus. Human cross-circulation, which was used successfully for a short period in the development of open cardiac surgery, has been supplanted by "heart-lung" machines. Methods of stopping and starting myocardial contractions are already being used to provide a quiet operative field, to minimize metabolic demands and to avoid the accumulation of harmful metabolites in heart muscle.

An increasing number of both congenital and acquired cardiac lesions will undoubtedly be approached by open cardiotomy. The purpose of this and subsequent chapters is to discuss briefly the essential features and the diagnostic criteria of those lesions of the heart and great vessels upon which there is at present general agreement concerning the surgical treatment. The details of the surgical techniques used to correct or modify these lesions will be illustrated and discussed.

Inflammation of the pericardium may lead to a thick fibrous or calcific enclosure which interferes with the movements and, to a varying degree, the function of the heart. It is generally believed that tuberculous pericarditis is the antecedent of constrictive pericarditis in most instances although pericarditis due to pyogenic organisms must be considered to be the primary lesion in some cases. The high initial mortality of acute pericarditis due to these latter organisms leaves few victims to develop constrictive pericarditis.

Rheumatic fever is considered to be a rare cause of constrictive pericarditis. It has been noted that the small hearts with thickened constricting pericardium usually have normal valves. The large hearts with disease of the valves and adherent but not constricting pericardium, comprise a different disease entity.

Constrictive pericarditis may develop within a few weeks if there have been heavy deposits of fibrin as in pneumococcal or staphylococcal pericarditis. Tuberculous pericarditis may behave similarly but more often years may elapse before constriction occurs. Undoubtedly the vigorous use of specific therapy for tuberculous pericarditis will decrease the number of patients with constrictive pericarditis of tuberculous origin.

Organization following hemopericardium has been shown to be responsible for

constrictive pericarditis in a small number of patients. However, in many patients with constrictive pericarditis a specific cause cannot be demonstrated. This has been true in almost half the cases in some series of autopsy reports in this disease.

Constrictive pericarditis is usually a disease of young people, most of whom are between the ages of 10 and 40 years. The average age of 100 patients in one series was 25 years. Males are affected twice as frequently as females. A history of previous pericarditis may be helpful in the diagnosis but more often no history of antecedent disturbance can be elicited. Symptoms develop gradually. There is exertional dyspnea, fatigue, and abdominal swelling; these early complaints may be accompanied or followed by edema of the legs. In many patients swelling of the legs develops after the appearance of the ascites. Emphasis has been placed on Beck's triad in the diagnosis of constrictive pericarditis, namely, a small quiet heart, a high venous pressure, and ascites.

Constrictive pericarditis interferes with diastolic filling thereby decreasing cardiac output. Systole is not hampered. Cardiac output can only be increased on exercise by increase in heart rate, not by increase in diastolic filling as occurs normally. The systemic and pulmonary venous pressures are increased as is the total circulating blood volume. As a result there is pulmonary congestion, hepatomegaly, ascites, and edema of the lower extremities which may be moderate and appear late.

The peripheral veins are distended throughout both systole and diastole due to the high venous pressure. This venous distension is greater than that seen in congestive heart failure and does not show the diastolic collapse or systolic engorgement of tricuspid insufficiency. Enlargement of the liver due to engorgement may cause epigastric pain. The liver edge is usually smooth, rounded and slightly tender. The enlarged liver in patients with constrictive pericarditis does not pulsate as it does in the presence of tricuspid insufficiency. Ascites, however, may obscure the hepatic findings.

The heart is small and quiet; there is minimal precordial activity. The tones are distant and scarcely audible. No murmurs are heard in most cases. Pleural effusion is common particularly on the right side. The peripheral pulses lack normal tension and fullness. The systolic pressure is usually low and decreases further with inspiration. A narrow pulse pressure ranging from 15 to 25 mm. of mercury and a normal diastolic pressure are usually found.

The venous pressure in the arm veins is sharply and persistently elevated to 300 to 400 mm. of water, the normal range being 50 to 150 mm. of water. Hepatomegaly, ascites, and edema usually are present when the venous pressure exceeds 250 mm. of water.

Cardiac catheterization has shown elevated pressures in the superior vena cava, the right auricle, right ventricle, and in the pulmonary circulation. Circulation time is prolonged.

Fluoroscopic examination discloses a normal or slightly enlarged cardiac shadow which appears to be fixed in position. Pulsations are extremely limited. Roentgen kymography accurately records the limited cardiac motion and corroborates the conclusions reached on the basis of the fluoroscopic examination; it may even suggest which regions are severely restricted. Roentgenograms, particularly in the oblique and lateral projections, may show calcification in the pericardium. Liver function tests may be abnormal in the presence of hepatic congestion and resulting fibrosis.

Constrictive pericarditis must be distinguished from congestive heart failure, cirrhosis, and other causes of ascites. A thorough trial of medical management aids diagnosis, as most patients with constrictive pericarditis do not respond well to these measures. Their failure to respond emphasizes the necessity for surgical treatment. Ascites and pleural effusion should be reduced by aspiration if necessary prior to pericardiectomy.

PERICARDIECTOMY

Weill's prediction in 1853 ". . . surgery will one day deliver the heart from the shell which strangles it" was first successfully accomplished in America by Churchill in 1928. Subsequent important contributions by Holman, Blalock and Burwell, and Harrington have further established pericardiectomy as a sound procedure.

The selection of the optimum time for operation is always important but may be difficult. In patients without any evidence of systemic response to infection and in whom only the mechanical effects of constriction are found, operation may be performed as soon as maximal benefit from medical preparation has been attained. On the contrary, it may be necessary to withhold operation for many months until fever, tachycardia, and elevated sedimentation rate subside. Calcification is evidence of a chronic process. Because of the high incidence of tuberculous etiology anti-tuberculous therapy should be part of the medical management despite lack of evidence of the causative agent.

Once a patient has had the maximal benefit from management with digitalis, salt restriction, mercurial diuretics, and removal of pleural and peritoneal fluid accumulations, surgery should be done. General anesthesia given through an endotracheal tube is used. Provision for adequate blood replacement should be made.

The choice of incision varies considerably. A vertical midline incision with splitting of a segment of, or the entire sternum in its midline has been used. A trans-section of the sternum in conjunction with intercostal incisions as required to provide adequate exposure has been favored by some. Either approach (Fig. 153) will provide adequate exposure which will permit a wide resection of pericardium anteriorly from the caval regions on the right to the pulmonary veins on the left.

If median sternotomy is used, it may be possible to free the pericardium from the pleura without opening either of the pleural cavities. Should a rent in the pleura occur, it may be closed by sutures.

The most important step in pericardiectomy is finding the proper cleavage plane. It should be sought over the anterior ventricular surface near the apex and away from the coronary vessels (Fig. 153). Injection of 1 per cent procaine solution into the pericardium may aid in the detection of the proper cleavage plane by hydrostatic pressure. The procaine may also allay arrhythmias.

The pericardium is mobilized by sharp and blunt dissection (Fig. 154). Special care must be exercised when the pericardium is being separated from the thin auricles and from the ventricles in the region of the coronary vessels. As the pericardium is freed, part of it may be excised but a flap should be preserved to cover any opening inadvertently made in an auricle or a ventricle. In areas where the pericardium is densely adherent it may be necessary to leave discs or strips of it undisturbed. The risk of removing every bit of pericardium may be much greater than the advantage to be gained by doing so.

The left phrenic nerve must be identified and protected as the dissection approaches the superior pulmonary vein on that side. The right phrenic nerve is less likely to be damaged but it should be sought as the dissection frees the superior vena cava.

In addition to the excision of the pericardium anteriorly and laterally, excision of its diaphragmatic portion is also indicated. In fact, this part of the pericardial sac is usually the thickest and most contracted, probably because of the gravitation of irritating exudate to this dependent position as suggested by Holman. That portion which encroaches on the inferior vena cava must certainly be excised to relieve or prevent constriction of that vessel.

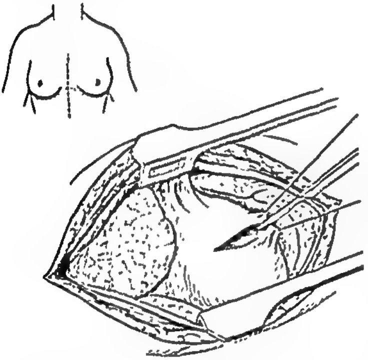

FIG. 153.

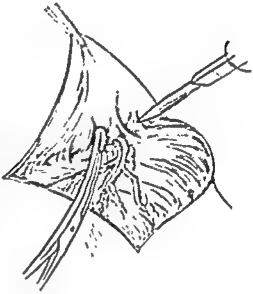

FIG. 154

FIG. 153.—Insert shows choice of incisions suitable for exposure necessary to accomplish adequate resection of pericardium in constrictive pericarditis. The detailed drawing shows the method for finding the proper line of cleavage between pericardium and epicardium.

An effective pericardiectomy is followed by prompt reduction in venous pressure and decrease in ascites and hepatomegaly. Persistence of ascites usually means an inadequate pericardiectomy. Beck has stated, "Partial operations yield partial results. . . ."

The results of the surgical treatment of constrictive pericarditis must be considered on the basis of a large series rather than the individual case.

Holman, in 1949, summarized the results of pericardiectomy in 265 patients. Approximately 45 per cent were considered cured. Fifteen per cent were improved. The spectacular results in about half of patients treated surgically must be contrasted with the obvious hazards as indicated by a mortality rate of approximately 25 per cent, one-third of these patients having died during the operation while the

remainder have succumbed during the postoperative period. Comparable results have been reported in 1956 in 78 patients.

QUESTIONNAIRE

1. What is the most common infection leading to constrictive pericarditis?
2. Constrictive pericarditis is found most frequently in which sex and age group?
3. List the main symptoms of constrictive pericarditis. Is their order of appearance helpful in the diagnosis?
4. Describe the altered cardiovascular physiology in constrictive pericarditis. What physical findings are explained on this basis?
5. What are the results of cardiac catheterization and venous pressure studies in constrictive pericarditis?
6. What are the radiographic findings in this disease?
7. What medical and surgical measures are indicated?
8. What should be the goal in surgical treatment and how is it accomplished?
9. What are the complications of pericardiectomy?
10. What results may be expected in properly selected cases treated by adequate surgery?
11. What are the causes of persistent ascites after pericardiectomy?

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CHAPTER 21

SURGERY OF THE HEART AND GREAT VESSELS—PATENT DUCTUS ARTERIOSUS

By John L. Keeley

The physiologic responses to the presence of a patent ductus arteriosus have been fully appreciated for decades. Halsted in 1910 called attention to the similarity between the effects of this lesion and those of a peripheral arteriovenous fistula. Holman studied 28 cases of patent ductus arteriosus and his interpretations of the effects of this lesion and types which include those with a reversal of the shunt are well described in an article published in 1925.

Graybiel, Strieder, and Boyer made the first surgical attempt to close a ductus complicated by bacterial endarteritis without success. Gross in 1938 accomplished the first successful closure by ligation of the ductus and pointed out that it seems just as reasonable to close such a fistula as it is to close a peripheral arteriovenous communication, a procedure already shown to be beneficial. He subsequently introduced the technique of division and closure of the ductus to prevent re-establishment of the shunt, a possibility when ligation in continuity is employed.

In fetal life the unexpanded lungs offer resistance to the flow of blood through them and the greater part of the blood from the right ventricle passes into the systemic circulation through the patent ductus arteriosus. After birth the lungs expand, pulmonary resistance decreases and the systemic pressure becomes relatively higher. The flow of blood through the ductus is reversed, flowing from the aorta to the pulmonary artery (Fig. 155). This shunt causes a murmur. It is thought that the pressures in the two circuits subsequently become equal and the ductus closes functionally long before an anatomical obliteration occurs. The murmur may disappear if no significant flow passes through the ductus arteriosus.

The obliteration of the ductus begins at the pulmonary artery end and is usually complete by the second month of life, at which time the murmur subsides. Spontaneous closure of the ductus may be due to some peculiar behavior of its tissue as it is not kept open by the relative pressures of the two circulations nor does its importance as a channel seem to have significance as it closes even in those patients in whom it is essential to life.

Patency of the ductus arteriosus after the age of two years represents an abnormality whether it is isolated or is associated with some other abnormality such as pulmonary atresia, in which instance it is the main path by which blood reaches the lungs.

The amount of blood shunted through a patent ductus arteriosus has been determined in some cases to be 20 to 70 per cent of the blood expelled from the left ventricle. This increases the work of both ventricles but the greater load is obviously on the left side of the heart as long as a left to right shunt persists.

In adult life, and occasionally at an earlier age, the increased volume of blood passing through the pulmonary vessels leads to pulmonary vascular sclerosis and

increased resistance to flow of blood through the pulmonary vascular bed. Right ventricular hypertrophy then develops. Eventually the pressure in the right ventricle equals or exceeds that in the left ventricle. This change in pressures causes a second reversal of the shunt which at first is partial but finally becomes complete. Then venous blood from the right ventricle reaches the aorta through the ductus. It causes cyanosis of the left side of the face, left arm, the lower part of the trunk and legs (Fig. 156). The eventual reversal of the shunt, plus the increasing hazards and technical difficulties of operation in older patients, the possibility of endarteritis of the ductus and blood stream infection with streptococcus viridans, justify recommendations for early surgical obliteration of the ductus.

Patent ductus arteriosus is two to three times more common in females. It is first discovered, in many patients, during a routine physical examination. This means that in most patients it does not cause marked physiological disturbances until the third or fourth decades. If the ductus is small symptoms are minimal but dyspnea, palpitation and decreased exercise tolerance occur when the ductus is large. Growth may be retarded in such patients.

The most characteristic finding is a so-called "machinery" murmur, harsh and rasping, best heard in the second interspace just to the left of the sternal border. It is louder in the recumbent position and has a systolic accentuation. The murmur continues throughout diastole because diastolic aortic pressure exceeds diastolic pulmonary artery pressure. Thus the flow of blood from the aorta to the pulmonary artery also occurs during diastole.

The systolic phase of the murmur is transmitted to the neck vessels and can also be heard high in the intrascapular area. In infancy only the systolic phase may be heard, but later systemic diastolic pressure rises and blood then flows through the ductus, both in systole and diastole. In addition to the murmur there is a thrill palpable in the same area as the maximal intensity of the murmur in about half of the cases and, like the murmur, it is more pronounced in the recumbent position. There is widening of the pulse pressure increasing it over the average normal of about 40 mm. of mercury. With a large ductus the pulse pressure may be so great that peripheral signs suggesting aortic insufficiency may be present.

Chest *x*-ray studies disclose the most common findings to be prominence of the pulmonary artery as indicated by a projection laterally from the left heart border just below the aortic knob (Fig. 157), and increased vascularity in the lung fields. Aortic pulsations transmitted to the pulmonary artery through the ductus cause the "hilar dance" which, incidentally, can also be seen in some patients with auricular septal defects.

The electrocardiogram usually indicates hypertrophy of both ventricles. In older patients there may be left axis deviation but if evidence of right axis deviation is found pulmonary stenosis, tetralogy, or, in the third decade and later, a reversal of the ductus flow must be suspected.

It may be said that with the current over-all mortality from surgical closure of the patent ductus arteriosus of approximately two per cent, probably all patients with this defect should be operated upon. The exceptions are those patients with reversal of the shunt, but careful screening may indicate that even some of these should be explored as to the possibility of closing the shunt. Blood stream infection with streptococcus viridans is an indication rather than a contraindication for operation. It can usually be brought under control with intensive antibiotic therapy prior to operation.

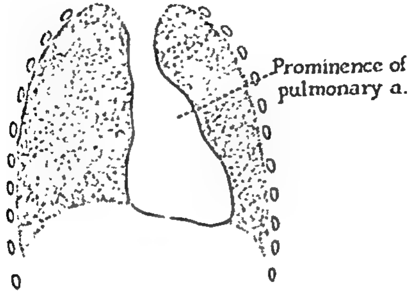

FIG. 157.—Contour of cardiac silhouette in uncomplicated patent ductus arteriosus. Cardiac enlargement is slight. The outstanding feature is the prominence of the pulmonary artery.

DIVISION AND CLOSURE OF A PATENT DUCTUS ARTERIOSUS

The currently acceptable surgical procedure in patent ductus arteriosus is division and closure by suture of the divided ends of the ductus. Simple ligation may be indicated in rare instances, but ligation in continuity can be followed by recanalization. Anesthesia with a tight fitting mask may suffice, but we are firmly convinced that an endotracheal tube is preferable as it will insure an adequate airway at all times, make controlled respiration possible, and provide for aspiration of bronchial secretions.

The chest is opened by a left intercostal incision usually in the third or fourth intercostal spaces. The anterior submammary approach through the third intercostal space with division of costal cartilages is usually satisfactory for children, but in teen-age and adult patients a standard lateral thoracotomy incision through the fourth intercostal space provides better exposure and allows adequate room for dealing with complicated technical problems and difficult ducti when the aorta must be mobilized in order to provide control should hemorrhage occur.

The pleura is incised posterior to the phrenic nerve. The ductus is easily found by locating the thrill which is usually maximal in a finger-tip sized area immediately overlying the ductus. The projection of pericardium can often be identified by the transmitted systolic impulse to pericardial fluid. Both the pericardial projection and pericardial fat are dissected off the ductus (Fig. 158). The vagus nerve, then the recurrent laryngeal nerve are identified, the latter requiring protection from trauma during the dissection, division, and closure of the ductus.

As soon as a sufficient length of the ductus has been freed (Fig. 159) a piece of heavy silk or narrow umbilical tape is passed behind the ductus. Traction can then be made, exposing more of the posterior surfaces and thus permitting sharp dissection under good visual guidance. A sufficient length of ductus should be exposed to provide adequate cuffs for secure closure of the divided ends. An inadequate length of ductus permits only narrow cuffs to protrude from the clamps. Even the slightest retraction of the ends, if they are short, may result in insecure closure by suture.

The results of operation include a rise in diastolic pressure if it has been low, slower pulse rate, disappearance of the murmur, and some decrease in heart size if enlargement has been present. The increased efficiency of the circulation leads to improved general health, alterations in personality traits, and in children frequently an accelerated rate of growth.

QUESTIONNAIRE

1. When is a patent ductus arteriosus normally present and what purpose does it serve?
2. What process appears to bring about spontaneous closure of a patent ductus arteriosus?
3. What is the eventual result of an uncorrected patent ductus arteriosus?
4. What is the chief objection to ligation as a method of interrupting flow through the patent ductus arteriosus?
5. What is the most characteristic physical finding? What are the other findings of significance?
6. What postoperative complications of surgical correction of a patent ductus arteriosus may occur?
7. What is the present mortality in the surgery of patent ductus arteriosus?
8. In which patients with patent ductus arteriosus is operation contraindicated?

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CHAPTER 25

SURGERY OF THE HEART AND GREAT VESSELS—COARCTATION OF THE AORTA

By John L. Keeley

Coarctation is the term used to describe a congenital obstruction to the lumen of the aorta which may vary in degree, extent and location. The most common type is that which occurs just distal to the origin of the subclavian artery in close relation to the attachment of the ligamentum arteriosum or ductus arteriosus. It almost completely occludes the lumen by a shelf-like inward protrusion of the media

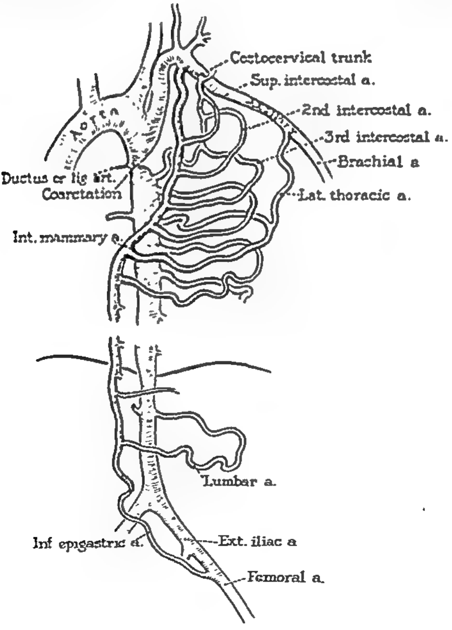


Fig. 163.—Diagram showing the typical location and form of coarctation of the aorta and the collateral circulation around it.

murmurs may be due to aortic regurgitation. Continuous murmurs heard over the back may be due to the volume of blood passing through collateral vessels in this region.

Electrocardiographic studies may suggest a coexisting lesion or aid in evaluating myocardial damage due to the hypertension. The diagnosis can be made in 98 per cent of cases on the basis of blood pressure levels and pulsations in the extremities aided by an *x*-ray study of the chest.

Surgical correction of coarctation of the aorta by resection of the obstructed segment and end-to-end anastomosis was first done by Crafoord in Sweden and Gross in the United States in 1945. The success of this procedure is attested by the decrease in the mortality from 20 per cent in 1950 to less than 10 per cent at present. The use of homologous aortic grafts to restore continuity after excision of a longer segment was described by Gross in 1951. Retrograde angiography through a brachial artery to demonstrate the site and extent of the coarctation has been done in many cases prior to the use of grafts. It is no longer necessary because the operation for the correction of coarctation should not be started without a graft or prosthesis.

The average age at death in patients with coarctation is about thirty years. One-fourth reach a normal life expectancy without difficulty, but three-fourths die in their early years in about equal numbers from three main causes: rupture of the aorta, superimposed bacterial endocarditis and the hypertensive state leading to cerebrovascular accidents or cardiac failure.

Because of the hazard which an uncorrected coarctation represents and the current surgical mortality which is approximately 10 per cent, it is generally agreed that operation should be recommended for all patients with coarctation who do not have serious valvular defects or myocardial damage. These two complications are more frequently encountered in the fifth and sixth decades. Their presence rather than the age of the patient contraindicates correction of the coarctation.

OPERATION

Because the greatest hazard in cardiovascular surgery is hemorrhage, ample blood for replacement must be available. In addition to needles or cannulæ usually placed in both ankle veins for rapid transfusion, a sterile setup for direct intra-aortic injection of blood should be available in the event of sudden massive hemorrhage. Provisions for determining blood loss by weighing used sponges and measuring the amount of blood aspirated from the operative site are the best guides to quantitative replacement.

Endotracheal anesthesia is used routinely. Hypothermia (30° C.) lowers the metabolic demands in all tissues and provides a factor of safety during operations for coarctation. It is generally conceded that the blood supply to the spinal cord is extremely variable. Should it be decreased during correction of a coarctation by temporary or permanent closure of collateral vessels, the lowered metabolic requirement of the tissues of the spinal cord cooled to 30° C. can be met. Hypoxia of the spinal cord which may result in neurologic defects ranging from mild sensory disturbances to paraplegia can thus be avoided. Hypothermia, therefore, is recommended in all operations in adults with coarctation and in younger patients who appear to have inadequate collateral channels.

RESECTION OF THE COARCTATION

The patient is placed on his right side and a pad is placed under his right chest wall so that his weight does not rest on his shoulder. An incision through the fourth intercostal space may be used in young patients but in the late teens and in adults control of bleeding from the tortuous intercostal vessels and their branches is more easily accomplished when a rib is removed and the relatively avascular rib bed is incised. If necessary, the posterior ends of a rib or two above or below the initial approach may be divided together with the related intercostal bundles. This provides the exposure necessary to correct the coarctation safely and to deal with complications should they arise. It is possible, in young patients, to spread the ribs wider and division of ribs may be unnecessary in those instances. Because of the large collateral vessels in the chest wall, *most of the blood loss usually occurs during the thoracotomy* unless hemorrhage is due to errors or accidents in the dissection, excision, and repair of the coarcted area.

Mobilization of the aorta above and below the coarctation and of the left subclavian artery should be done first. The dissection of those intercostal arteries which must be sacrificed or those necessary to permit mobilization of the aorta requires special care.

An intercostal artery, when torn, usually pulls out of its point of origin in the aorta leaving an opening rather than a stump of vessel which could be ligated. This accident is difficult to deal with but the opening can be closed by an "X" suture of silk in the aortic wall. The site of the coarctation is dissected last and the division of the ligamentum arteriosum or ductus arteriosus finally frees this area. It may be necessary to divide a bronchial artery arising from the aorta at this level.

When hypothermia is used, the blood pressure is usually significantly decreased. In patients with marked hypertension and friable vessels additional means to lower the pressure may be advisable to make the mobilization, excision, and anastomosis of the coarcted segment safer. Induced hypotension with one of the ganglion blocking agents adds a definite factor of safety in the dissection and manipulation of the aorta, particularly in relation to that portion above the coarctation.

Lowering the pressure decreases possible damage to the aortic wall when clamps are in place as the pulse wave "pounds" the aortic wall against the upper clamp. This is particularly true in patients with severe hypertension and atherosclerosis. Suitable clamps should be applied above and below the area to be resected in a manner which leaves sufficient cuff of aortic wall for accurate approximation by suture. Clamps should avoid the proximity of the stumps of intercostal arteries which may be sheared off by slight slipping of the clamp.

Excision of the coarcted area should result in openings in the aorta of equal size, the edges of which are free of intercostal artery stumps which complicate suturing (Fig. 165). Enough of the coarctation must be excised so that the anastomotic line does not constrict the aortic lumen. If the ends cannot be approximated without undue tension due to inelasticity, a graft or prosthesis must be used. Other indications for the use of either graft or prosthesis are shown in Figure 166.

The steps in suturing the ends of the aorta after excising the coarcted area are shown in Figure 167. The eversion of the edges is started by one or two interrupted mattress sutures in the most inaccessible segment of the proposed anastomosis. A continuous mattress suture is used to complete the anastomosis. Figure

168, A shows an anastomosis made in this manner. Figure 168, B shows an anastomosis made with a simple over-and-over continuous suture. The suture material is 5-0 silk swedged on fine round needles. In younger patients, a combination of interrupted and continous sutures may be used to avoid any constricting effect and to provide for dilatation of the anastomotic site as the caliber of the rest of

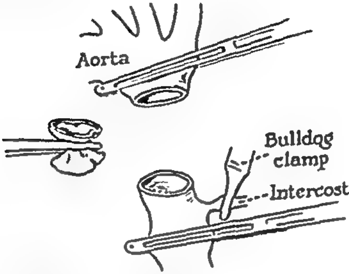


FIG. 165.—After mobilization of the coarctated segment, sufficient length is excised to provide normal caliber at the site of the anastomosis. A bulldog clamp temporarily occludes an intercostal artery.

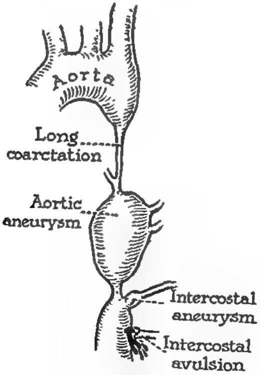


FIG. 166.—Four indications for use of a homograft or prosthesis are shown.

the aorta increases with growth. If it is necessary to use a graft or prosthesis, the same suture techniques can be employed. A replacement segment no longer than necessary will avoid angulation or bowing and establish the continuity of the aorta in a straight line (Fig. 169).

An alternate method of dealing with a long segment of coarctation by use of the left subclavian artery is shown in Figure 170. This procedure may be used if no graft is available or in *young patients with a subclavian artery of suitable size.* There is a risk of gangrene of the left arm if this method is employed in adults.

The suture lines in *all types of repair* may be tested by first releasing clamps on intercostal arteries which have been temporarily occluded. The distal aortic clamp is next released. Finally the proximal clamp is opened slowly to void any sudden large alterations in the peripheral resistance leading to fall in blood pressure

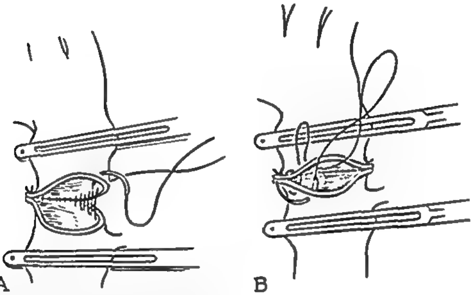
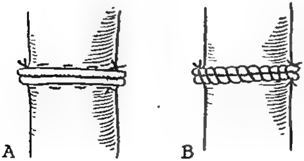


Fig. 167.—*A*, Eversion of the aortic edges may be initiated by one or two interrupted mattress sutures in the least accessible segment of the proposed anastomosis. The posterior half is then approximated by a continuous mattress suture.
B, Approximation of the anterior edges by a continuous mattress suture completes the anastomosis.



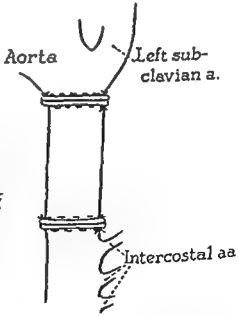


Fig 168 Fig. 169

Fig 168—Two types of anastomoses are shown, one made with the everting mattress suture and the other with the simple over-and-over stitch.

Fig. 169—Diagram showing a homograft used to restore continuity. It is placed under slight tension which prevents bowing and kinking.

and insufficient coronary filling. Gradual release also avoids the rapid circulation of metabolites which may have developed in areas of stasis. The goal should be release of the proximal clamp without any significant changes in the pulse or respiratory rate or in the blood pressure. To accomplish this may take several minutes. If suture lines or stitch holes leak, an additional stitch or two may be necessary but pressure with gauze for a few minutes usually suffices. The mediastinal pleura is closed over the site of repair to favor an early perivascular support of coagulum to the anastomotic area. The standard closure of the chest wall plus intercostal catheter drainage is done.

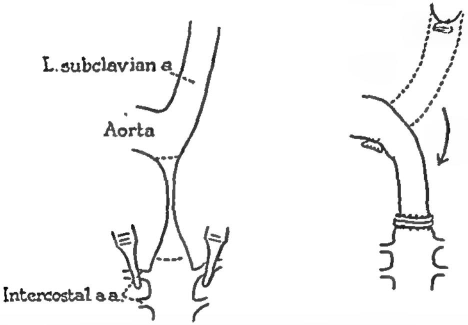


FIG. 170 —An alternate method of repairing the aortic defect. The left subclavian artery in many patients with coarctation compares favorably with the caliber of the aortic arch and descending aorta. Adequate collateral circulation to the left arm must be present to justify this method.

Some serious but fortunately rare complications may occur. Obviously hemorrhage from the suture line may be fatal. In rare cases, in the older adult group, disturbances in the blood supply to the spinal cord may result in serious neurologic disturbance in the legs varying from motor and sensory paralysis of limited extent to paraplegia. Postoperative infections with resistant organisms may be fatal. Other complications are those of thoracic operations in general such as atelectasis, pulmonary edema, pneumonitis and residual chest pain of varying degrees.

The blood pressure in the upper extremities may remain somewhat elevated in adult patients but usually drops within two to three weeks in the younger group. Return to normal levels occurs in about 90 per cent of cases. Blood pressure levels in the legs may reach normal values promptly or within a few weeks. In some instances the pressures may remain lower than normal.

The results to date are extremely encouraging. Although the use of grafts, prostheses, hypothermia and induced hypotension have been factors in the reduction in mortality from the early figure of 20 per cent to the current one of about 10 per cent, the most important factor in further reduction will be earlier diagnosis and treatment in the optimum age group which is from ten to twenty years. Homologous grafts have now been used for over six years. There appears to be a tendency for mild degrees of calcification to develop in the region of the graft as

shown by roentgenographic studies but aneurysm formation has not been detected. There is no doubt that operation in the older age groups has prolonged life by preventing or delaying the occurrence of aortic rupture, cerebrovascular accidents, or cardiac failure.

QUESTIONNAIRE

1. Describe the morphologic characteristics of a typical coarctation of the aorta.
2. In which patients should coarctation be suspected?
3. What are the main collateral channels which carry blood beyond the level of the coarctation?
4. What is the prognosis in patients with uncorrected coarctation of the aorta?
5. How is the diagnosis of coarctation of the aorta made?
6. What are the *x*-ray and electrocardiographic findings in coarctation?
7. What are the contraindications to its surgical correction?
8. Name the two most serious complications in the surgery of coarctation.
9. How does [illegible] n?
10. [illegible] prosthesis?
11. [illegible] in correction of coarctation of the aorta?
12. What is the optimum age group for operation in coarctation of the aorta?

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CHAPTER 26

SURGERY OF THE HEART AND GREAT VESSELS—VASCULAR COMPRESSION OF THE TRACHEA AND ESOPHAGUS

By John L. Keeley

As the primitive vascular system in the embryo is transformed into the normal vascular system of man, one is impressed with the endless possibilities for variation. Nevertheless this complex and ever changing network eventuates in the aortic arch and its major branches in a persistent and uniform manner with few exceptions. In the past, the variations which occur were carefully noted in autopsy reports and were of interest because they could be explained on an embryological basis.

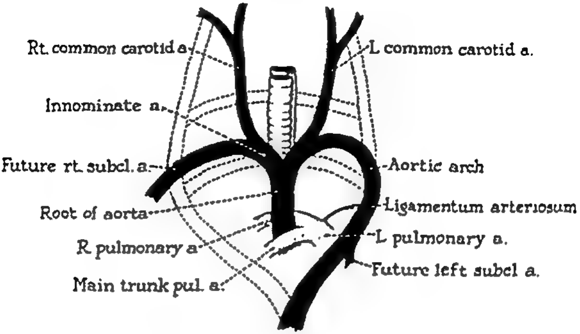
In 1931 while performing an autopsy on a five-month-old baby, Gross found a ring of vessels surrounding the trachea and esophagus. The constriction caused by the ring was thought to be sufficiently severe to account for the wheezing respiration present since birth and the dysphagia of more recent origin. It seemed reasonable that if this vascular ring could be divided the release of the air and food passages would be followed by the subsidence of respiratory and swallowing symptoms.

Fourteen years later Gross was successful in performing such an operation on a four-month-old infant in whom roentgenographic studies showed compression of both the trachea and esophagus. Wheezing which had been present since birth was relieved.

With the establishment of surgical treatment as a sound procedure in patients with constriction due to vascular rings, the detection of these abnormalities becomes of more than academic interest.

The pioneer embryologists and, more recently, interested clinicians have described the formation of the vascular network, its transformation into the normal arrangement and its abnormalities in great detail. One may say briefly, however, that the embryonal aortic arches and their adjacent vascular channels are arranged so that they encircle the primitive anlage from which the trachea and esophagus are derived (Fig. 171).

Right aortic arch is a common vascular abnormality. It is formed from the right fourth aortic arch. It is associated with a thoracic aorta which may follow one of three courses: (*a*) The thoracic aorta may continue distally on the right side. This arrangement is found in from 20 to 25 per cent of patients with Tetralogy of Fallot. (*b*) The thoracic aorta may cross to the left, indent the esophagus posteriorly, then pass distally to the left of the esophagus. (c) The thoracic aorta may pass to the left and upon reaching a point behind the esophagus form a bulge or diverticulum, then return to the right side and continue distally. From the bulging retroesophageal portion of the aorta, either the ductus arteriosum, ligamentum arteriosum, or the left subclavian artery may arise.



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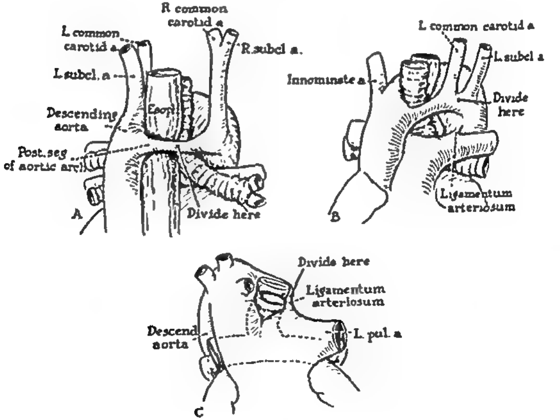

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The double aortic arch is the abnormality which causes the most severe constriction of the trachea and esophagus. It is not associated with cardiac anomalies as a rule. It is possible because of the failure of involution of the right aortic arch which normally contributes to the formation of the right subclavian artery. The double aortic arch is formed by the right aortic arch which swings behind the esophagus from the right to join the left aortic arch which passes to the left in front of the trachea. The right (posterior) component joins the left (anterior) component to the left of the trachea and esophagus to form the thoracic aorta, which in most patients with double aortic arch passes distally in the left side of the chest.

Three types of double aortic arch appear to be the most frequently encountered. One variety has a small right (posterior) component and a larger left (anterior) component (Fig. 172, *A*). A more common type is that in which this relationship is reversed, the smaller component being anterior (Fig. 172, *B*). The third arrange-

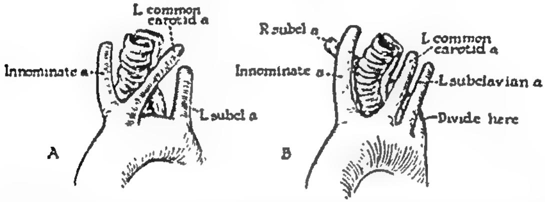

Fig. 173.—Two types of vascular compression due to the course of major branches of the aortic arch. (*a*) The left common carotid artery causes compression of both trachea and esophagus. (*b*) The anomalous origin of the right subclavian artery causes esophageal compression.

ment consists of a double aortic arch with a right descending aorta. The left (anterior) arch is the smaller one in this situation and reaches behind the esophagus to complete the encirclement posterior or to the right of the esophagus. Partial obliteration is encountered occasionally in the left (anterior) arch but only rarely in the right (posterior) one.

In some patients having a right aortic arch, a constricting ring may be formed by the *ligamentum arteriosum* which extends from the pulmonary artery, passes posteriorly to the left and then behind the trachea and esophagus to join the right aortic arch (Fig. 172, *C*). In addition to the constriction caused by the ligamentum arteriosum, a taut left subclavian artery arising from the junction of the right aortic arch and right descending aorta may further compress the esophagus on its posterior surface.

The anomalous origin of the innominate and left common carotid arteries causes them to press on the trachea to a variable degree (Fig. 173, *A*). The right subclavian artery may arise as the last branch of the aortic arch and pass in front of the trachea, behind the esophagus or between the two strucutres (*Fig. 173, B*).

SYMPTOMATOLOGY

In patients with a double aortic arch, mild or moderate hesitation in swallowing due to esophageal compression is often present. The symptoms of tracheal com-

pression, however, are the ones that attract much more attention. The respiratory rate is increased, there is a struggle for adequate aeration, the accessory muscles of respiration are used, and there is intercostal and suprasternal retraction. Wheezing may be heard usually without the stethoscope. Breathing is of the crowing type and stridor, both inspiratory and expiratory, is present. There may be a history of recurrent respiratory infections.

Tracheal compression is increased by swallowing, and taking food therefore increases the respiratory distress. Infants often assume a position in which the neck is hyperextended. This appears to put extra traction on the trachea and improves the airway. Flexion of the neck in these patients promptly interferes with the airway.

The symptoms may not be quite as severe with a right aortic arch and a left ligamentum arteriosum and they may come on later in childhood than the symptoms which are seen in double aortic arch. In patients with anomalous origin of the innominate artery or of a left common carotid artery, the compression is usually on the left side of the trachea and causes only mild respiratory symptoms. The esophagus is free from compression and no difficulty in swallowing is experienced.

In most patients whose right subclavian artery arises as the most distal branch of the otherwise normal aortic arch and passes behind the esophagus, no compression of importance occurs. In some instances, however, the vessel may be taut enough to interfere with peristalsis and hesitation in swallowing may result. Since the compression is high in the esophagus in this situation, regurgitation may occur and aspiration may cause respiratory distress. Respiratory symptoms do not occur otherwise as there is no tracheal compression.

DIAGNOSIS

The most important step in the diagnosis is an awareness of the possibility of tracheal or esophageal compression or both in a patient, usually an infant or child, who has had respiratory distress, recurrent respiratory infection, or difficulty in swallowing. Roentgenograms of the chest in routine and special projections may

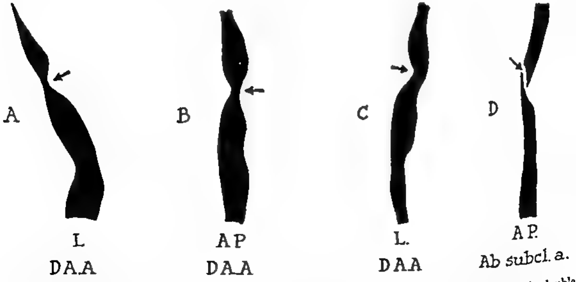

FIG 174.—Diagrams show esophagrams in the lateral and anterior-posterior projections in double aortic arch (D A A.) and the anterior-posterior vein in aberrant subclavian artery.

show pneumonitis or evidence of decreased excursion on inspiration and expiration. Widening of the superior mediastinum in the anterior projection may suggest a vascular ring. Tracheograms using a little Lipiodol will permit demonstration of irregularities due to external compression of the trachea. These are not likely to be severe except in cases of vascular rings due to double aortic arches.

Bronchoscopic examination is done to rule out primary disorders of the respiratory tract, such as the laryngeal papillomas or webs, congenital cysts, endobronchial webs and nonopaque foreign bodies. A barium swallow will show interference with peristalsis and indentation of the esophagus. The type of defect may suggest the caliber of the compressing structure (Fig. 174). The large rounded indentation of the esophagus may suggest that the larger component of a double aortic arch is posterior, whereas a sharp, narrow indentation suggests that it may be due to the ligamentum arteriosum or to a narrow posterior arch. In addition, the indentations due to the components of a double aortic arch are more likely to be at right angles to the direction of the esophagus whereas the aberrant or anomalous branches of the aortic arch often make diagonal impressions.

SURGICAL THERAPY

Careful study of patients showing symptoms of tracheal or esophageal compression should suggest the abnormality which is most likely to be encountered. Most of them can be approached from the left side. However in some patients in whom defects of the left latero-posterior surface of the esophagus are due to the larger left (anterior) aortic arch, it may be necessary to approach the smaller anomalous right (posterior) arch from the right side.

General anesthesia is used. An endotracheal tube must be used in these patients to provide an airway which will not be narrowed or closed by changing the position of the head or by the manipulation of the mediastinal structures during the course of extensive dissection so necessary in the search for that portion of the constricting ring which may be divided safely.

The approach to the superior mediastinum may be made through the third left intercostal space unless the roentgenographic studies suggest that the smaller and perhaps partially obliterated posterior arch may be more accessible from the right. The more recent experiences with transverse incisions in the anterior chest wall in which the sternum is divided and the incision is carried into adjacent intercostal spaces make the choice of the side on which the incision is begun of less importance.

The thymus gland can be dissected free and the greater part of it may be removed. The aortic arch and its major branches are carefully freed and encircled with tapes for their subsequent manipulation. Accurate identification of each channel must be made so that the type of abnormality can be verified. Two procedures are followed in the surgical treatment. In one, a nonessential part of a vascular ring or a nonessential aberrant artery may be divided. The second procedure consists of traction by sutures to pull a vital vascular channel toward the anterior chest wall and relieve pressure, particularly on the trachea until that structure becomes large and firm enough to resist pressure from the vascular abnormality.

There are specific procedures for the most frequently encountered variations. In the double aortic arch with a large anterior limb the smaller posterior limb may

be divided at a point where circulation to the carotid and subclavian vessels will not be impaired (Fig. 172, *A*). In the patients having a double aortic arch with a large posterior limb and a small anterior limb, surgical cure can be obtained by the complete division of the smaller anterior aortic arch (Fig. 172, *B*). In patients with a double aortic arch which is associated with a right descending aorta, both the anterior arch which encircles the trachea and esophagus on the left side and the ligamentum arteriosum which forms a second constriction at a slightly lower level may be divided with release of the trachea and esophagus.

In patients with a right-sided aortic arch and left ligamentum arteriosum, the constriction may be relieved by dividing the ligamentum arteriosum (Fig. 172, *C*). Division of the left subclavian artery which may come off of this retroesophageal portion of the aorta may also be necessary to relieve constriction. The collateral circulation around the shoulder girdle usually is sufficient in children to provide adequate circulation to the arm after the division of the subclavian artery, but it may be tested by temporary occlusion of the vessel.

The trachea can be compressed by an innominate artery which arises more distally from the aortic arch than normal. In order to return to the right side it must cross the trachea and in doing so may compress it to some extent. Mobilization of this vessel and its attachment to the anterior chest wall will relieve the trachea of compression from this source. Occasionally the left carotid artery arises from the aortic arch in common with the innominate artery or very close to it (Fig. 173, *A*). In this situation, the left carotid artery then must cross the trachea and in so doing may cause enough compression to produce symptoms. Some ingenuity may be necessary to devise means of lifting the aortic arch and the important branches away from the front of the trachea to relieve the pressure from this source.

There is of course no standard procedure which can be recommended in all cases of vascular compression of the trachea and esophagus. Occasionally the right subclavian artery comes from the aortic arch as the last branch and crosses to the right behind the esophagus (Fig. 173, *B*). In this situation, the right subclavian artery is doubly ligated and divided near its origin from the aortic arch, again testing the collateral circulation around the shoulder joint prior to the ligation and division of the vessel.

The complications of surgical correction of vascular compression of the trachea and esophagus are those of thoracotomy plus those associated with the dissection, manipulation and ligation of large arterial channels. Apparently the good results of surgery for vascular compression of the trachea and esophagus are permanent. No reports of follow-up studies of these patients have been encountered.

QUESTIONNAIRE

1. Describe the formation of a double aortic arch.
2. What history, symptoms or findings would make you suspect vascular compression of the trachea and esophagus?
3. How is the diagnosis established?
4. What other abnormalities must be considered in the differential diagnosis? How are they ruled out?
5. Discuss the surgical management from the standpoint of anesthesia, incision, and specific procedures for the different types of anomalies.
6. What may one expect from surgical treatment?
7. What are the complications of surgical treatment for vascular compression of the trachea and esophagus?

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CHAPTER 27

SURGERY OF THE HEART AND GREAT VESSELS—MITRAL STENOSIS AND REGURGITATION

BY JOHN L. KEELEY

THE END results of rheumatic fever may consist of myocardial damage and valvular deformities in varying degrees and proportions. The mechanical effects of mitral stenosis and regurgitation have long been recognized and in many patients overshadow any disability which can be attributed to disturbances in myocardial function.

In order to overcome the effects of mitral stenosis, attempts to enlarge the mitral orifice have been considered for some time. Sir Lauder Brunton of England suggested a surgical approach to the problem in 1902. An attack on a stenotic pulmonary valve was made by Doyen in 1914. Tuffier, in 1915, dilated a stenotic aortic valve. However, it was not until 1925 that Souttar digitally dilated a stenotic mitral valve. He reached it through the auricular appendage. The result was good, but this promising approach attracted little attention.

In 1925 Cutler and Beck using a transventricular approach enlarged the stenotic mitral orifice by excising a portion of valve leaflet with a punch-like instrument called the cardiovalvulotome. Stenosis was thus replaced by regurgitation. Smithy, Boone, and Stallworth aroused renewed interest in this type of surgical treatment of mitral stenosis and reported their efforts with a new valvulotome in 1950 using both transventricular and transauricular approaches.

The great impetus to mitral valve surgery came late in the decade following 1940 when Bailey, Harken, and Brock made such tremendous contributions to the field. The enlargement of the stenotic valve, so-called commissurotomy, either by finger pressure or by specially constructed knives or both through a transauricular approach is now recognized as a well established surgical procedure and has given highly satisfactory results in a large proportion of properly selected cases.

The ideal patient for commissurotomy is one who has limitation of activity and exertional dyspnea but who is not incapacitated. There may be a history of systemic failure but it should have been transitory with prompt response to appropriate treatment. Pulmonary edema or paroxysmal nocturnal dyspnea suggest a better outlook than do bouts of systemic failure. History of embolic disturbances in patients with auricular fibrillation suggests the need for operation. Hemoptysis associated with attacks of coughing is related to pulmonary congestion and is usually not massive. It is an additional indication for operation.

The diagnosis of mitral stenosis can usually be made on physical examination The so-called opening snap of the stenotic valve is due to the adherent leaflets acting as a diaphragm which is set in vibration before the diastolic rumble is heard. The opening snap is not heard in cases where the chordæ tendineæ are adherent and thickened by long-standing inflammatory processes. In heavily calcified valves the opening snap is also absent and careful evaluation must be made as it

is unlikely that far advanced lesions such as the calcified ones can be sufficiently altered by surgery to cause significant improvement.

The diastolic rumble due to blood passing through the stenotic orifice from auricle to ventricle characteristically ends in a crescendo leading into the first sound which is due to the closure of the mitral and tricuspid valves. The normal apical "lubb" becomes snappy as the mitral valve closes in the presence of stenosis.

The roentgenogram shows enlargement of the left auricle and right ventricle and some pulmonary congestion. If the left ventricle is also enlarged, the patient is a poor candidate for mitral surgery. The electrocardiogram should show right ventricular hypertrophy and not many other significant changes.

Cardiac catheterization is not necessary to make the diagnosis of mitral stenosis. However, it has been useful in investigating the altered dynamics of mitral stenosis and evaluating the results of commissurotomy. Cardiac catheterization can also be used to prove the functional nature of symptoms in cases of mild mitral stenosis in which the typical murmur is the sole demonstrable abnormality.

Direct transmural catheterization of the left auricle is valuable in the selection of advanced cases for operation. It is accomplished by passing a long needle under fluoroscopic control through an interspace in the right paravertebral area in the direction of the central portion of the cardiac shadow. In patients with both mitral stenosis and insufficiency the relative importance of each has been assessed by left heart catheterization. Thus it is possible to select patients who can be helped by commissurotomy but who would ordinarily be considered unsatisfactory candidates for the operation.

The presence of mitral regurgitation is usually indicated by a loud systolic murmur maximal at or near the apex of the heart, that is, over the clinical mitral valvular area. The murmur is transmitted, as a rule, to the axilla and, if intense, may be heard over the entire precordium and in the left subscapular area. This systolic murmur usually occurs early in systole, becomes progressively more intense, then less intense as systole progresses. A systolic thrill, absent as a rule, may accompany the loudest systolic murmurs.

There is no satisfactory operative procedure at present for mitral insufficiency. Commissurotomy, while opening a stenotic valve, may also provide mobility to the valve leaflets permitting more effective closure of the valve and thus decrease or, rarely, completely correct regurgitation. In some patients regurgitation may be increased during commissurotomy and therefore patients with significant mitral insufficiency must in general be considered poor candidates for commissurotomy. A few patients will be benefited if the stenosis can be corrected without seriously increasing the insufficiency.

Patients with mitral stenosis under twenty years of age usually have enough latent rheumatic activity despite negative tests for its presence to warn against operation. Their symptoms are related to rheumatic myocarditis rather than to the mechanical effect of the stenotic valve in most instances. Patients between the ages of 30 and 45 years have the most favorable result from commissurotomy. They have enough myocardial reserve to benefit from the correction of the stenotic valve. The valve may still be a favorable one for commissurotomy in this period. While age is not a contraindication, the risk of operation increases with advancing years. Many patients in their sixties have been distinctly improved by relieving mitral stenosis. In general, the bigger the heart, the larger the left ventricle, the more chronic the fibrillation, the less possibility there is that the patient will be helped by operation.

THE TECHNIQUE OF MITRAL COMMISSUROTOMY

Anesthesia is induced, the trachea is intubated, and the patient is turned from the recumbent position to lie on his right side. A resilient but not completely compressible pad such as a bath blanket or two wrapped in two layers of inch-thick foam rubber is placed so that the right side of the chest, not the shoulder girdle, supports the patient. The arms are suitably supported and protected by soft pads.

The incision begins medial to the mid-clavicular line, passes under the breast area in either sex, curves posteriorly and upward around the angle of the scapula and reaches the level of the mid-point of the vertebral border of the scapula and half way between the vertebral border and the spinous processes. The latissimus dorsi, serratus anterior, and portions of the trapezius and rhomboid muscles are divided allowing the scapula to be retracted superiorly. The erector spinæ fascia is freed and retracted away from the third to the fifth ribs. By passing the hand under the scapula the second rib can be identified by the insertion of the middle and posterior scalenus muscles and the proper interspace, usually the fourth, is identified.

The contour of the chest and configuration of the heart may suggest an approach at a different level in some instances. The intercostal incision is made from the costochondral junction to the transverse processes of the spine. The internal mammary artery is usually only a finger's breadth from the edge of the sternum and can easily be avoided. Posteriorly, the intercostal artery and vein tend to leave the groove under the rib about 3 to 4 cm. from the transverse process and pass medially in the mid-portion of the intercostal space. The intercostal incision can be directed along the upper margin of the inferior rib to avoid the vessels in this region.

The ribs are spread gradually, the lung is retracted posteriorly, the pericardium is exposed, and palpation for thrills of diagnostic value is done. The pericardium is opened high enough to expose the pulmonary artery and this incision extends down to the diaphragm below. It is usually made anterior to the phrenic nerve although in some patients it may be advantageous to make the incision posterior to the nerve.

The exposed heart is examined for thrills at the base of the aorta suggesting aortic stenosis, and over the left auricle indicating mitral regurgitation. Palpation of the auricle and auricular appendage should be gentle to avoid the possibility of dislodging clots. If none is suspected, infiltration about the base of the auricle with one per cent procaine solution without adrenalin is done preparatory to placing the auricular purse-string suture. Infiltration with procaine may be unnecessary in many cases. Nevertheless we have found it effective in decreasing auricular irritability in cases where the installation of the purse-string suture was begun without it.

The purse-string suture, usually #2 white silk swedged on a needle, is placed about the base of the auricle without passing the needle through the full thickness of the auricular wall. It is then threaded through a Rumel tourniquet (Fig. 175). A suitably shaped auricular clamp is placed at the base of the appendage and closed only enough to prevent bleeding as the tip of the appendage is amputated. This tissue provides a biopsy to determine if there is histologic evidence of rheumatic activity. The opening should be just large enough to permit introduction of the finger (Fig. 176). The edges of the opening may be grasped with Mixter forceps to provide countertraction as the finger is introduced into the snug fitting opening

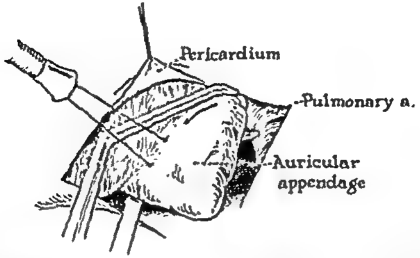

FIG. 175.—The pericardium has been incised. A purse-string suture has been placed at the base of the auricular appendage. The ends are led into a Rumel tourniquet. A non-crushing auricular clamp is in position.

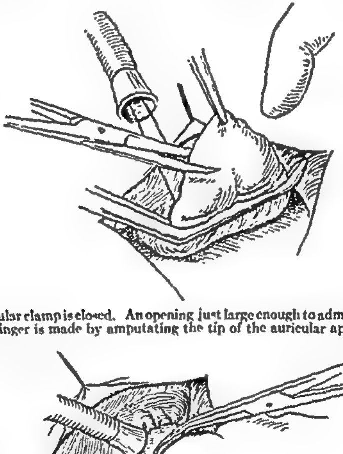

FIG. 176.—The auricular clamp is closed. An opening just large enough to admit the surgeon's index finger is made by amputating the tip of the auricular appendage.

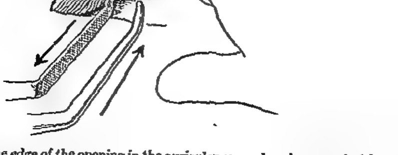

FIG. 177.—The edge of the opening in the auricular appendage is grasped with a Mixter forceps to provide countertraction as the finger is inserted and the auricular clamp is withdrawn. The tourniquet can be tightened if necessary if the finger does not fit the opening snugly.

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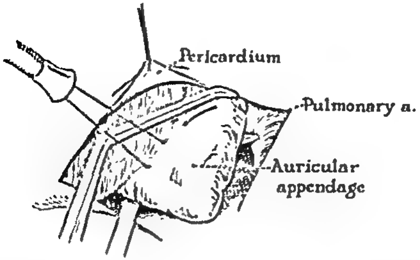

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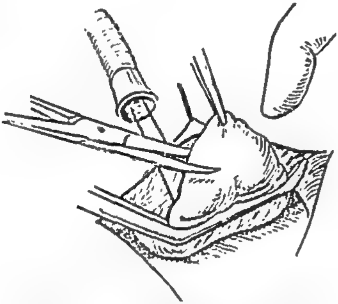
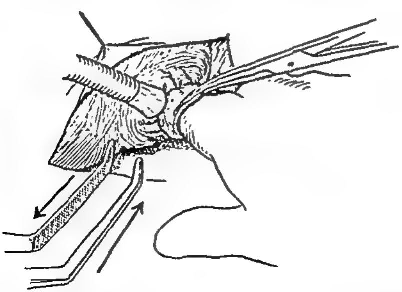


FIG. 176.—The auricular clamp is closed. An opening just large enough to admit the surgeon's index finger is made by amputating the tip of the auricular appendage.



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(Fig. 177). Some trabeculations of the appendage may need dividing to permit introduction of the finger.

A poor risk patient, a tiny valve opening, unsatisfactory cardiac function or hypotension may require rapid enlargement of the valve orifice either by finger separation of the commissures or incision of them with a suitable knife (Figs. 178, 179). In less urgent situations exploration of the auricle and of the valve surfaces may be done unhurriedly to detect clots, regurgitation, calcification, atrial septal defects, and the size and position of the valve orifice. One must avoid dislodging clots or calcific granules. Compression of the carotid vessels during manipulations in the presence of clots or calcification may decrease the possibilities of cerebral emboli.

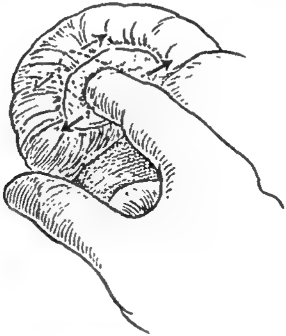


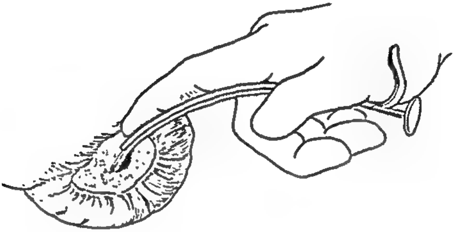
FIG. 178.—Arrows indicate direction of force to separate leaflets at anterior and posterior commissures.

The mitral valve leaflets may be compared to the pages of a new book; some will separate easily, others actually must be cut in order to avoid tearing into either page (valve leaflet). Detachment of chordæ tendineæ by either cutting or tearing may result in serious and sometimes fatal regurgitation. A limited increase in the size of the valve orifice without causing or increasing regurgitation is certainly preferable to a wide opening and a serious degree of regurgitation.

In order to study the effects of the mitral lesion on the dynamics of the circulatory processes pressure studies made in the pulmonary artery and in the auricle may be done by direct measurement with suitable apparatus before and after commissurotomy. A significant fall in these pressures, particularly the auricular pressure, after commissurotomy and while the systemic pressure remains unchanged usually indicates that marked clinical improvement may be expected.

The exact technique of separating the commissures requires orientation by touch alone. The necessary force to cause the separation must be carefully controlled to avoid tears in the valve leaflets, auricular wall, or chordæ tendineæ. Commissurotomy should be accomplished in a series of limited steps so that should regurgitation result manipulations can be stopped before it becomes severe.

Some patients have such limited cardiac reserve that the finger must not be kept in the valve orifice for more than a few beats as it obstructs the flow of blood. This leads to decreased cardiac output and hypotension, the most serious results of which are poor coronary filling and decreased cerebral circulation. Quite often, therefore, the enlargement of the valve opening must be done in a somewhat piecemeal manner allowing for correction of hypotension and improvement in cardiac action between short periods of intracardiac manipulation.



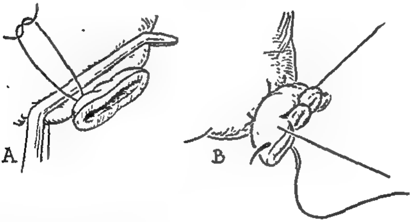


FIG. 180.—*A*, The auricular clamp has been applied as the finger is withdrawn. The first knot has been made in the purse-string suture.
B, Additional silk sutures reinforce the purse-string closure of the tip of the auricular appendage.

If the commissures resist digital separation, a suitable knife must be used (Fig. 179). The procedure is completed by withdrawing the finger and at the same time applying the auricular clamp. The purse-string suture is then tied and additional silk sutures are used to reinforce this closure (Fig. 180, *A* and *B*). The traumatized edges of the opening in the auricular appendage may be excised. The pericardium is irrigated with normal saline solution. Closely placed silk sutures approximate the edges at the level of the auricular appendage. Above and below this level the sutures are widely placed to permit gaping for the escape of pericardial fluid which may accumulate in the postoperative period. The tip of the lingula may be amputated to provide a pulmonary biopsy. A standard thoractomy

tube is brought out in the mid-axillary line through a suitable intercostal space, such as the eighth or ninth.

The lung is expanded and the chest is closed by means of pericostal sutures of catgut and continuous catgut sutures in the muscle, fascia, and subcutaneous fat. Silk is used to close the skin. The patient is then examined for the presence of peripheral pulses to detect evidence of peripheral embolization. As soon as the patient is sufficiently awake, voluntary movements are demonstrated to rule out the presence of any cerebral embolic disturbance.

Management in the postoperative period is largely medical, dealing principally with the management of cardiac function and treatment of a flare-up of rheumatic activity, the so-called post-commissurotomy syndrome, should it occur. The postoperative surgical problems are infrequent as a rule but include pain, atelectasis, empyema, pleural effusion and sometimes bleeding from the chest wall. Late arterial embolization is uncommon. The results of commissurotomy depend upon the proportion of disability due to the tight mitral orifice and to what degree this has been corrected. The presence of mitral regurgitation, myocardial damage, pulmonary fibrosis and involvement of other valves are the other major factors which determine the ultimate benefit.

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QUESTIONNAIRE

1. What is the etiologic background in most patients with mitral valve lesions?
2. What early surgical attempts were made to correct mitral stenosis? Were they different from those currently employed?
3. Describe the auscultatory findings in mitral stenosis. In mitral regurgitation.
4. On what clinical basis should mitral commissurotomy be recommended? In which patients should surgery be withheld?
5. Is cardiac catheterization necessary or helpful in selecting patients with mitral lesions for surgery?
6. Describe the technique of commissurotomy.
7. Is mitral regurgitation amenable to surgical correction at present?
8. What are the complications, operative and postoperative, of mitral commissurotomy?
9. Upon what factors do the results of commissurotomy depend?

CHAPTER 28

SURGERY OF THE HEART AND GREAT VESSELS—PURE PULMONIC STENOSIS

By John L. Keeley

Stenosis of the pulmonic valve as an isolated abnormality in an otherwise normal cardiovascular system has been considered a rare lesion. Recent reports, however, are recording an increasing number of patients with this anomaly. The introduction of a surgical approach for its correction by Doyen, Brock, Sellors, Swan and others has made the accurate diagnosis of this lesion most imperative. The results of its surgical correction can be among the most gratifying of any attained in treating intracardiac lesions.

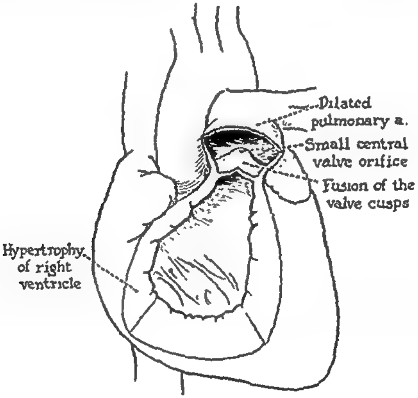

Fig. 181.—The main features of "pure" pulmonic stenosis are shown: the hypertrophied right ventricle, fusion of the valve cusps, a small central valve orifice, and dilatation of the pulmonary artery.

Normally, the pulmonic valve has three semilunar cusps. Congenital fusion of these cusps results in a funnel or dome-shaped valve with a central opening which may be no more than 3 mm. in diameter (Fig. 181). It opens into a pulmonary artery two to three times normal size due to post-stenotic dilatation. This is the so-called valvular type of pure pulmonic stenosis.

Narrowing of the right ventricular outflow tract or pulmonary conus of the right ventricle (Fig. 182) is called infundibular stenosis and is quite common in

tube is brought out in the mid-axillary line through a suitable intercostal space, such as the eighth or ninth.

The lung is expanded and the chest is closed by means of pericostal sutures of catgut and continuous catgut sutures in the muscle, fascia, and subcutaneous fat. Silk is used to close the skin. The patient is then examined for the presence of peripheral pulses to detect evidence of peripheral embolization. As soon as the patient is sufficiently awake, voluntary movements are demonstrated to rule out the presence of any cerebral embolic disturbance.

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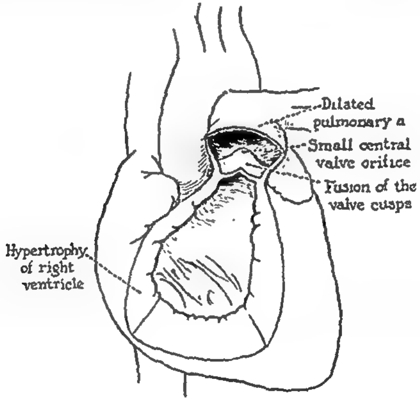

Fig. 181.—The main features of "pure" pulmonic stenosis are shown: the hypertrophied right ventricle, fusion of the valve cusps, a small central valve orifice, and dilatation of the pulmonary artery.

Normally, the pulmonic valve has three semilunar cusps. Congenital fusion of these cusps results in a funnel or dome-shaped valve with a central opening which may be no more than 3 mm. in diameter (Fig. 181). It opens into a pulmonary artery two to three times normal size due to post-stenotic dilatation. This is the so-called valvular type of pure pulmonic stenosis.

Narrowing of the right ventricular outflow tract or pulmonary conus of the right ventricle (Fig. 182) is called infundibular stenosis and is quite common in

tetralogy of Fallot but relatively rare as a single isolated anomaly. It is accompanied by a pulmonary artery of diminished rather than increased size as seen in the valvular type (Fig. 182). Both valvular and infundibular stenoses may be present in the same patient. The degrees of stenosis are quite variable. We have seen a woman of 45 years with pure pulmonary stenosis with a normal-size heart and minimal symptoms whereas a high grade stenosis can lead to heart failure in the early weeks or months of life. There is an increased incidence of pulmonary tuberculosis in patients with pulmonic stenosis presumably due to decreased pulmonary blood flow.

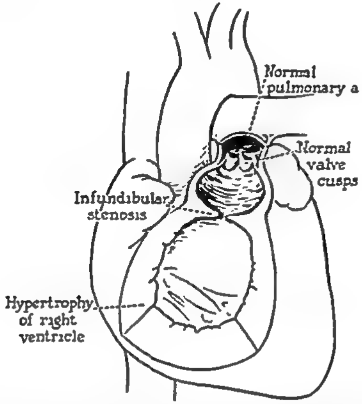

FIG. 182.—Infundibular stenosis. The constriction occurs in the outflow tract below the valves and forms two chambers. The wall of the right ventricle is hypertrophied. The pulmonary artery is normal or diminished in size in contrast to the post-stenotic dilatation of pure valvular stenosis.

The diagnosis of pure pulmonic stenosis is suggested by a history of easy fatigability and exertional dyspnea out of proportion to the extent of effort. Squatting may occur but is not nearly as frequent as seen in tetralogy. There is no cyanosis unless right auricular pressure becomes elevated enough to shunt venous blood through a patent foramen ovale.

A loud harsh systolic murmur in the second and third left interspaces is present. It is often accompanied by a thrill. Diastolic murmurs are rare. The second pulmonic sound may be increased with slight stenosis or decreased with severe stenosis. A split-second sound at the base associated with other findings of pure pulmonic stenosis suggests an infundibular rather than a valvular type of pulmonic narrowing. The systolic pressure usually does not exceed 100 mm. of mercury and the pulse pressure is narrow.

The hemogram is normal and the arterial oxygen saturation is within normal limits. The circulation time may be normal but often is prolonged. The electrocardiogram shows right ventricular hypertrophy, right axis deviation, or right

bundle branch block. Tall P waves indicate right auricular enlargement, changes in the T waves suggest myocardial strain in severe cases. Roentgenographic studies show enlargement of the right ventricle and diminished pulsations in the pulmonary artery and its branches.

There is usually decreased vascularity in the lung field. An enlarged pulmonary artery is an encouraging finding as it is highly suggestive of the valvular type of stenosis which is technically easier to correct (Fig. 181). Enlargement of the right auricle occurs only in impending right heart failure as a rule.

Cardiac catheterization is essential in establishing the diagnosis and also to determine right ventricular pressure as a basis for recommending operative treatment. It is likewise necessary to distinguish valvular from infundibular stenosis as the surgical approach may vary in the treatment of these two types. The oxygen content of blood samples obtained during cardiac catheterization reveal no intracardiac shunt.

The normal systolic pressure of the right ventricle is from 20 to 30 mm. of mercury. When it reaches 70 to 80 mm. of mercury in cases of pure pulmonic stenosis, operation should be performed. When the pressure reaches 200 mm. of mercury, operation is urgently needed to forestall impending cardiac failure.

The cardiac catheter attached to a device for continuous recording of pressure, shows a sharp fall in pressure as the tip of the catheter passes through the valve orifice in the valvular type. Since the orifice is so small the catheter itself causes increased obstruction and the pulmonary artery pressure is further decreased at this time. In cases of infundibular stenosis a drop in pressure occurs as the catheter passes into the constricted outflow tract often constituting an intermediate chamber "walnut size" as viewed externally. A further drop in pressure is recorded as the catheter passes through the valve into the pulmonary artery.

Angiocardiography is usually not necessary to establish the diagnosis but visualization of the stenotic area can corroborate the catheterization data suggesting the type of stenosis. The dye is delayed for a time in the right ventricle and pulmonary artery showing them both to be increased in size; decreased vascular markings in the peripheral lung fields may be demonstrated.

SURGICAL TREATMENT

The relief of obstruction in the valvular, infundibular or combined types can be accomplished by either open or closed methods. The original procedures were done through an antero-lateral approach in the fourth left interspace dividing costal cartilages above and below to provide the necessary exposure. In the presence of right ventricular hypertrophy, the anterior wall of the right ventricle can be rotated into view from its position under the sternum by traction medially on the flap after a]-shaped incision has been made in the pericardium. In one adult patient this was not possible due to previous tuberculosis on the right side leading to adhesion and fixation of the pericardiopleural surfaces. It was necessary to rongeur away almost two-thirds of the width of the sternum to expose the right ventricle for a safe procedure. No disability resulted from the sternal defect.

Once the chest is opened in pulmonic stenosis, the cardiac action may deteriorate rapidly and extremely prompt enlargement of the valve orifice may be necessary to save the patient's life. A knife with a long narrow blade may be plunged through the right ventricular wall to incise the valve as the first move once the pericardium is opened. A cataract knife is a suitable instrument for this purpose in infants.

The large pulmonary artery in the valvular type is soft and easily compressible and the tense, almost rigid, funnel or dome-shaped valve projecting into its lumen is easily palpated as is the accompanying jet of blood coming through the tiny opening. These findings plus the absence of an infundibular chamber confirm the diagnosis of the valvular type.

Purse-string or mattress sutures are placed through almost the entire thickness of the wall of the right ventricle, care being taken to avoid any coronary artery

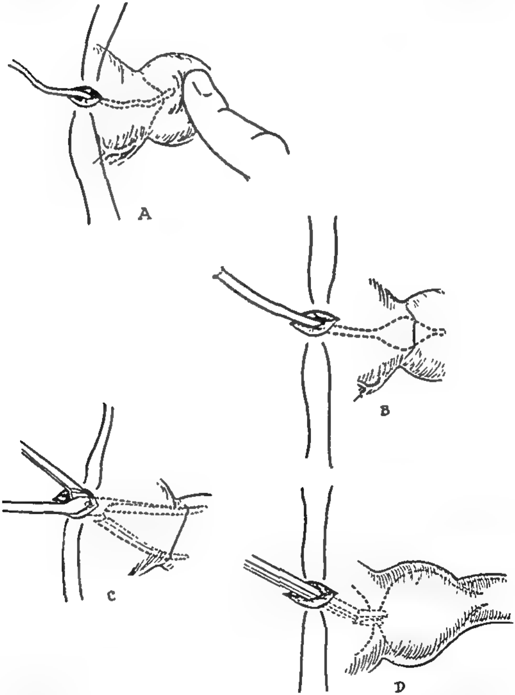

Fig 183 — *A*, A suitable probe is passed through the wall of the right ventricle and locates the valve orifice.

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branches in the area. An incision with a knife is made within the sutured area to approximately the same depth as the sutures. The valve area is examined by a probe pushed through the remaining uncut muscle fibers. An ordinary uterine sound is a satisfactory probe and sometimes the opening through the valve is so small that an instrument of this size almost completely blocks it (Fig. 183, *A*). The probe is withdrawn and the valve is split with Brock knives or some suitable modification thereof (Fig. 183, *B*).

Although many complicated dilators have been designed to stretch the valve orifice after incising the valve with knives, ordinary curved Kelly forceps serve admirably (Fig. 183, *C*). The hinge rests in the myocardial incision and spreading the jaws and handles does not cause serious blood loss. Pressure studies should be made at the operating table to prove that the valve opening is sufficiently large to allow the right ventricular pressure to return to normal or to as low a level as the greatest valve opening possible will permit.

Infundibular stenosis can be approached in the same manner; a Brock rongeur (Fig. 183, *D*) is used to bite away the edge of the stenotic area to relieve the obstruction in the outflow tract. The results in these cases are less satisfactory than those attained by the Brock procedure in the valvular type. In rare instances when both valvular and infundibular stenoses are present the latter may require correction before the valve can be opened. The myocardial incision is closed with a minimal number of silk sutures which exclude the endocardium.

Postoperative catheterization studies have shown that valves treated by incision and subsequent instrumental dilatation have been either simply stretched, inadequately opened or have not remained open. For this reason Swan, using hypothermia, approaches the pulmonary artery and the right ventricle through a transverse incision across the sternum opening the fourth interspace on the right and the third interspace on the left. With body temperature reduced to 30° C. the circulation can be interrupted for a period of seven or eight minutes providing the patient was adequately oxygenated at the beginning of this time. The azygos vein is ligated, the cavæ are occluded by traction tapes and a clamp is placed across the pulmonary artery distal to the site where it is to be opened. The opened pulmonary artery empties in a few heart beats and the valve can be incised under direct vision to the pulmonary arterial wall and a portion of the tip of the valve may be excised (Fig. 184). An adequate opening without regurgitation can thus be achieved. The edges of the arteriotomy are caught in a suitable clamp, circulation is re-established and the artery is closed by a continuous mattress suture with fine silk.

Should there be infundibular stenosis, an incision directly into it through the ventricular wall may be made and the obstructing tissue rongeured away (Figs. 185 and 186). Since circulation may be interrupted for a limited time only, the correction of the infundibular type must be done rapidly as it may be necessary to remove several bites of tissue and the ventricular wound must be sutured to close it instead of applying a clamp as can be done to close the wound in the pulmonary artery quickly. These procedures under hypothermia can be done equally well through a midline sternum-splitting incision which may avoid entrance into the pleural spaces, a technique used effectively by Julian. The simplicity of the Brock procedure and the excellent clinical response which follows in a high percentage of cases make it a more popular procedure at present than the more complicated and hazardous direct approach using hypothermia and interruption of circulation.

The pericardium is closed with silk sutures at points which allow escape of

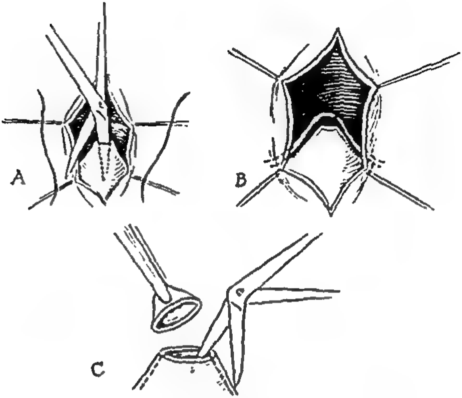

Fig. 184.—*A*, Open method of correcting pure pulmonic stenosis. Accurate and adequate incision of the stenotic valve leaflets may be achieved in this method.

B, Incisions have been carried to the point of attachment of the valve leaflets providing maximal opening.

C, A portion of the valve may be excised if desirable. The remaining cone can then be divided to the point of attachment as in *B*.

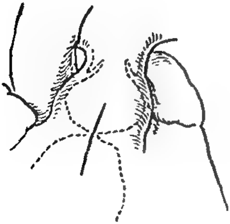

Fig. 185

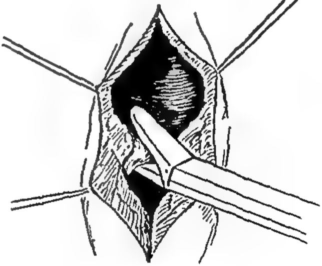

Fig. 186

Fig. 185.—Open method of correcting infundibular stenosis. Ventriculotomy into the constricted area exposes the obstruction. This alone may correct a significant portion of the obstruction.

Fig. 186.—Excision of the ridge of muscular tissue may be accomplished by a rongeur as shown or by scissors or scalpel.

pericardial fluid in the postoperative period. Closure of the chest wall is done in the usual manner. If the sternum or costal cartilages are divided transversely, special attention to their approximation and stabilization is necessary. The other incisions usually heal without difficulty as a rule. Bed rest is indicated for a period of two to three weeks, depending upon the postoperative electrocardiographic changes to allow complete healing of the myocardial wound. The improvement in exercise tolerance is quite striking and after a period of weeks or months the electrocardiographic evidence of right ventricular hypertrophy may disappear in the more favorable cases and cardiac catheterization may show return of pressure to near normal values.

QUESTIONNAIRE

1. Compare the valvular and infundibular types of stenosis as to auscultatory catheterization and operative findings.
2. Under what circumstance may cyanosis occur in pure pulmonic stenosis?
3. What findings are of significance in the *x*-ray study of the chest in pure pulmonic stenosis?
4. What have been the results of the closed operation for pulmonic stenosis?
5. What is the advantage of open operation; the disadvantages?

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CHAPTER 29

SURGERY OF THE HEART AND GREAT VESSELS—AORTIC STENOSIS AND REGURGITATION

By John L. Keeley

The correction of mitral stenosis by commissurotomy has suggested the application of somewhat similar methods for the relief of aortic stenosis. While there have been some favorable results following dilatation of the valve, in general it is a more hazardous and less satisfactory procedure by far than are similar attacks on the mitral valve.

Aortic stenosis may be congenital but most often it is due to either rheumatic fever or arteriosclerosis. In the older age groups most stenotic valves are calcified to some extent and in these it may be impossible to establish the etiology with certainty.

Two general types of deformity of stenotic aortic valves have been described. One has a fusion of all three cusps leading to a small centrally placed triangular opening with rigid edges. This opening can neither open nor close and regurgitation of course is always present in this type. In the other type, the two anterior leaflets fuse and a bicuspid valve results. There is then a thickened, immobile leaflet anteriorly and a more normal mobile leaflet posteriorly.

Some distinction may be made between the valves which are calcified and those which are not. The noncalcific type of aortic stenosis is usually due to rheumatic fever and is associated with aortic insufficiency and mitral stenosis. It is seen usually before the age of 50 years. Males predominate slightly. The cusps agglutinate and may lead to a minute triangular orifice. The calcific type usually also occurs predominantly in males (from 50 to 80 per cent); many live to an advanced age despite the narrowing.

The normal circumference of the aortic valve is 7.5 cm. It is often reduced to 2 cm. or less in aortic stenosis. The aortic valve area is reduced from the normal *of approximately 3 square cm. to .5 square cm. in fully developed aortic stenosis.* The critical area, that is, the area when symptoms of congestive failure usually appear, is .5 square cm. for aortic stenosis alone and 1.5 square cm. for combined aortic stenosis and aortic insufficiency

Pure aortic stenosis effects males predominantly. Duration of life after the onset of congestive failure is shorter and there is a higher incidence of syncopal attacks than is usually seen in patients with stenosis and some degree of regurgitation or in those having multivalvular disease.

The symptoms of congestive failure may first call attention to the disease. Chest pain may be present. It is most commonly referred to the right side of the chest or begins in the right side. Classic angina is related to stenosis rather than to coronary disease. Cerebral ischemia may cause syncope, faintness, focal paralysis, odd mental behavior, weakness, and flashes of light. The symptoms and physical

findings depend upon degree of stenosis and the functional capacity of the left ventricle.

Both the systolic and diastolic blood pressures vary. Systolic hypertension may be present; the diastolic value may be low or zero, depending upon the presence of associated regurgitation. In stenosis there is a harsh basal systolic murmur, maximal in the second right intercostal space. A coarse thrill is often associated with the louder murmurs. Auricular fibrillation is not common as in mitral stenosis or when mitral stenosis is associated with aortic stenosis. Calcification of the aortic valve can be seen in careful fluoroscopy but rarely has it been demonstrated on films. Sosman and Wosika conclude from their studies that calcification of the aortic valve means a severe degree of aortic stenosis.

Electrocardiographic and roentgen evidence of left ventricular hypertrophy is usually present. If the left ventricle is not enlarged aortic stenosis is mild.

Left heart catheterization has yielded some information concerning the degree of stenosis as indicated by the gradient across the valve. In the stage of compensation the left ventricular pressure during systole rises and may be normal during diastole, that is 10 mm. of mercury or less. In left ventricular failure the diastolic pressure rises as the left ventricle does not empty completely. In aortic stenosis the aortic pressures may be 120/86, which is essentially normal, while a left ventricular pressure of 220/30, for example, might be present. The gradient across the valve of 100 replaces the normal gradient which is but a few millimeters of mercury. In moderate stenosis the gradient ranges from 20 to 50 mm. of mercury, and in severe stenosis it can reach 50 to 100 mm. of mercury or more.

The cardiac output in aortic stenosis may be normal during the resting period. It may rise with exercise in the less severe cases but not as much as it should. In severe cases cardiac output may be decreased at rest and if this cannot be increased by exercise it indicates a very poor prognosis.

Sudden death is not unusual in aortic stenosis particularly in patients who have had vertigo, syncope or angina. It is due to coronary occlusion, myocardial infarction, severe myocardial ischemia or cerebral anemia. Bacterial endocarditis is rare in aortic stenosis.

The average age at death in aortic stenosis was 65 years in Mitchell's series. In patients with both aortic stenosis and aortic regurgitation it was 52 years. The average duration of life after the onset of congestive failure is shorter in patients with aortic stenosis only than it is in those who also have regurgitation.

Twenty per cent of patients with severe clinical manifestations have so much calcium in the valves that they cannot be changed much by any surgical maneuver. It is generally felt that an operation should be considered where the gradient was over 50 mm. of mercury as determined by left heart catheterization. In general, an operation is not done in the asymptomatic group but operation is done in that group with fatigability and with angina or syncope and those having exertional dyspnea. Operation is contraindicated in patients with marked aortic insufficiency, complete heart block or advanced right heart failure. Patients beyond the age of 50 years are not regarded as good candidates, regardless of the other findings which might be favorable.

Since patients with aortic stenosis do not have the reserve to survive repeated bouts of congestive failure, commissurotomy should be considered without waiting for repeated episodes of failure.

To date there have been three methods of aortic commissurotomy. In the

first, a dilating instrument reached the valve area through a transventricular approach. More recently a trans-aortic route has been used. In this method a dilating instrument is inserted through a purse-stringed opening in the ascending aorta or through a "sleeve" attached to the edges of the aortotomy. The sleeve permits the introduction of a finger for examination and dilatation much as the auricular appendage does. The third method is the open one. It has been accomplished by interruption of the circulation under hypothermia or with the use of a cardiac bypass made possible by a pump oxygenator.

The surgical treatment of aortic stenosis resembles that of mitral stenosis because in both the object is threefold: to correct stenosis; to avoid causing or increasing regurgitation; and to restore function of the valve. The problems and hazards are much greater, however, in aortic stenosis. Valves with minimal calcification recover some degree of function when the leaflets are separated. Study of heavily calcified aortic valves at autopsy casts doubt on any optimistic opinion concerning the restoration of function in them.

AORTIC COMMISSUROTOMY

The technique of transventricular treatment of aortic stenosis is as follows.

Preparations for entering the chest rapidly to treat cardiac arrest or ventricular fibrillation must be complete as these conditions are prone to occur in patients with aortic stenosis as anesthesia is induced. Rapid induction with intravenous agents is in order. An endotracheal tube is inserted and thereafter respirations are controlled.

The chest is opened through the left fifth intercostal space. The innominate and left common carotid arteries are isolated through an incision in the pleura midway between the phrenic and vagus nerves. Arrangements to occlude these vessels by tapes or suitable tourniquets are necessary because of the high percentage of aortic stenotic valves which are calcified. The brain must be protected from calcific emboli during manipulations, which might dislodge calcium particles from the valve.

The pericardium is incised widely just anterior to the phrenic nerve to provide access to the root of the aorta and to the anterolateral surface of the left ventricle. One per cent procaine solution is infiltrated into the myocardium in an avascular area of the apex. In this infiltrated area, a purse-string of heavy silk arranged in a triangular fashion is put in place taking fairly deep bites. Within this triangle a puncture wound almost through the thickness of the myocardium is made. Through this wound, the aortic dilator is introduced as the purse-string controls bleeding around the instrument.

The aortic valve orifice is small and therefore a smooth-tipped wire guide is used with some dilators. A two-or three-bladed dilator separates the valve leaflets or may tear the leaflets at other points (Fig. 187). The character of the valve leaflets, the firmness of their fusion and the degree of calcification determine the extent to which the stenosis can be altered.

The dilator is opened to a predetermined dimension to avoid using excessive force which might tear the aortic wall instead of separating the thick leaflets attached to it. The result obtained will be determined by the size of the opening made in the valve, the avoidance of causing or increasing regurgitation and the mobility of the separated leaflets.

The dilator is withdrawn, the purse-string is tied and additional sutures as necessary to provide hemostasis are used. The pericardium is closed loosely with widely separated sutures. Effusion can drain through the incision into the pleural cavity but after two or three days this route usually becomes sealed off. If there is any question concerning pericardial effusion pericardiocentesis through the zyphocostal route should be done.

The retrograde approach to the aortic valve utilizes an anterior incision through the third intercostal space on the right or a mid-line sternum splitting incision. Isolation of the innominate and left common carotid arteries as described for the transventricular approach is done. A portion of the presenting wall of the ascending aorta is grasped in a suitable occluding clamp.

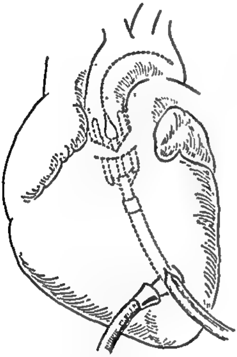

Fig. 187

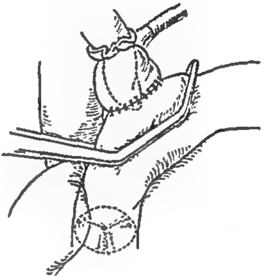

Fig. 188

Fig. 187.—The transventricular approach for the relief of aortic stenosis. A wire guide leads the dilating instrument to the tight aortic orifice. The opening in the ventricle near the apex is controlled by the purse-string leading into the Rumel tourniquet.

Fig. 188 —The aortic valve may be approached from above. This shows a "sleeve" attached to the edges of an incision into a segment of the aorta which has been isolated by a suitable clamp. A purse-string controls the opening into this sleeve.

A small caliber dilator can be introduced into the aorta through an incision in a purse-stringed area. A better approach by far is that which attaches a sleeve of pericardium or prosthetic material to the edges of an incision in the aorta so that a chamber is provided. Through this appendage digital or instrumental examination or dilatation of the valve can be accomplished (Fig. 188). Location of the valve orifice by following the systolic jet to its source aids in guiding the dilator.

Dilatation is done while the head vessels are occluded to avoid cerebral emboli. The results of dilatation can be evaluated by re-inserting the finger. The procedure is terminated by replacing the clamp on the aorta, removing the sleeve and closing the aortic incision with a suitable suture, usually a continuous mattress suture which everts the edges of the aortic incision.

The advantages of the retrograde approach are avoidance of damage to the ventricular wall, fewer disturbances in rhythm and the ability to evaluate the condition of the valve and the results of dilatation by digital examination.

As in the surgery of pulmonic stenosis, open methods are being investigated and have been subjected to clinical trial in a few patients. This has been done with hypothermia or extracorporeal circulation. It is reasonable to expect that the separation of fused valve leaflets can be done safer and more precisely under direct vision.

The physiologic effects of aortic stenosis are more serious than those found in other valve lesions. The surgical approach to this less accessible valve is more hazardous. It is therefore expected that the mortality is comparatively high (about 25 per cent) and the good results rather infrequent. Most appraisals of results have been made on clinical grounds alone. Obviously studies of the pressure gradient across the valve would form a better measure of what is accomplished in the surgical treatment of aortic stenosis.

AORTIC INSUFFICIENCY

Aortic insufficiency is usually due to rheumatic fever or lues. Unusual causes are bacterial endocarditis, trauma, or congenital factors. It is predominantly a disease of males and before the age of 35 years can usually be ascribed to rheumatic fever. The aortic cusps are shortened, their edges become thick, the annulus dilates, all of which contribute to the incompetence of the valve. Some degree of stenosis may be associated with aortic insufficiency in rheumatic cases but in the luetic cases aortic stenosis does not develop as the cusps separate rather early and are not adherent as in rheumatic heart disease.

Patients with aortic insufficiency may remain completely compensated for many years due to the efficient left ventricle and competent mitral valve. This is in contrast to the disturbance seen in mitral disease where a thin-walled left auricle becomes dilated and where passive congestion of the pulmonary vascular bed occurs.

Eventually the enlarged heart in aortic insufficiency may attract the patient's attention by its forceful beat, and pulsation of cervical or pharyngeal vessels may also be noticed. Dizziness due to postural hypotension occurs; angina is not common in uncomplicated aortic insufficiency of rheumatic origin, it is usually due to coronary sclerosis in older patients or to the narrowing of the coronary ostia in luetic cases.

Dyspnea on exertion is one of the earliest signs of left ventricular failure. Paroxysmal dyspnea, that is, a sudden attack occurring after exercise or more often during sleep or at least at rest lasts up to 15 minutes and is seen more frequently in luetic than in rheumatic aortic insufficiency.

Physical examination shows the apex beat extending out to the axilla and down to the sixth or seventh interspaces. There may be a systolic depression rather than outward thrust and diastolic pulsation may be present as the ventricle fills promptly due to the recoil from the aorta. There is a soft, high pitched diastolic murmur which is the most characteristic auscultatory finding. It starts immediately after the second sound and is best heard in the second right interspace or along the left border of the sternum in the third to fourth interspaces. The high pitch and the soft quality of this murmur make it difficult to hear many times; this is more apt to be the case in rheumatic than in luetic disease, where it is usually heard without

difficulty. Thrills are not usually present because of the high frequency of the murmur. An apical systolic murmur may indicate left ventricular failure as the left ventricle dilates and produces relative mitral insufficiency.

Peripheral signs are related to the low diastolic and high systolic pressures. The diastolic pressure is usually less than 50 mm. of mercury with a pulse pressure of 80 to 100 mm. or more instead of 30 to 50 mm., which is considered the normal pulse pressure. Corrigan's water hammer pulse is felt and is best appreciated when the arm is raised overhead. The pulse rate may be normal in compensated patients but moderate tachycardia (90 to 100 per minute) is frequent.

The vessels in the neck and the head vessels show a marked pulsation and there may be systolic movements of the head. Inspection shows pharyngeal pulsations and capillary pulsation may be demonstrated in the mucus membrane of the lips by partially compressing them with a glass slide or in the nails by pressing the edge of the nail down (Quinke's sign). Hill's sign refers to much greater pressure in the femoral artery than in the brachial artery. Normally the femoral artery pressure is only 10 to 20 mm. of mercury greater than the brachial artery pressure but in aortic insufficiency this difference may be 60 to 100 mm. Hg.

Auscultation in the region of the femoral artery discloses a pistol shot sound; it is a booming sound and sometimes it is double (Traube's double sound). Duroziez's sign is really a double murmur. Pressure over the femoral artery causes the systolic component and the diastolic murmur is due to the aortic insufficiency.

The cardiac shadow is boot-shaped. The left border is enlarged downward and to the left causing an increased angle with the left auricular salient. The aortic knob is usually prominent and the aortic shadow extending to the right of the sternum is curved and prominent.

The electrocardiogram shows left ventricular hypertrophy early in the course of aortic insufficiency. Sinus rhythm is present, premature ventricular contractions are common and many patients show first degree *a-v* heart block.

SURGICAL TREATMENT

Two approaches have been made to correct or to eradicate some of the effects of aortic insufficiency. Bailey has attempted to place a nylon sling beneath the coronary vessels which when tightened "cinches" the aortic annulus approximating the valve edges and thus decreasing the regurgitation. The difficulties in placing this sling and the results of its migration have caused it to be discarded.

Hufnagel has designed and used a plastic valve which is inserted into the descending aorta (Fig. 189). Studies have shown that blood in contact with methyl methacrylate does not clot. Therefore this material was chosen for the valve.

At the time the valve was first used clinically it was placed in the descending aorta just beyond the left subclavian artery for two reasons. The technique of placing it in the ascending aorta presented then insurmountable problems. More important, however, was the fact that the filling of the coronary vessels is dependent upon the diastolic recoil of the elastic aorta and great vessels and the amount of blood contained therein.

The Hufnagel valve corrects approximately 70 per cent of regurgitation when placed in the aorta just beyond the origin of the subclavian artery. This of course decreases the extent of the great vessels from which the "run-off" for coronary filling comes. The work load of the heart decreases and although coronary filling

is likewise decreased, it is decreased less than the decrease in work load. The over-all effect is one of distinct clinical improvement in 60 per cent of properly selected patients. The hospital mortality for the operation is about 25 per cent.

General anesthesia through an endotracheal tube is used. The descending aorta is exposed in the same manner as for correction of coarctation of the aorta. A sufficient number of intercostal vessels are tied and divided to permit the mobilization of the aorta for a distance of 8 to 10 cm. The nylon rings are put in place about the aorta at the upper and lower regions of the mobilized segment. Aortic clamps are applied; first the proximal one, then the distal one. The aortic wall is divided for about two-thirds of its circumference.

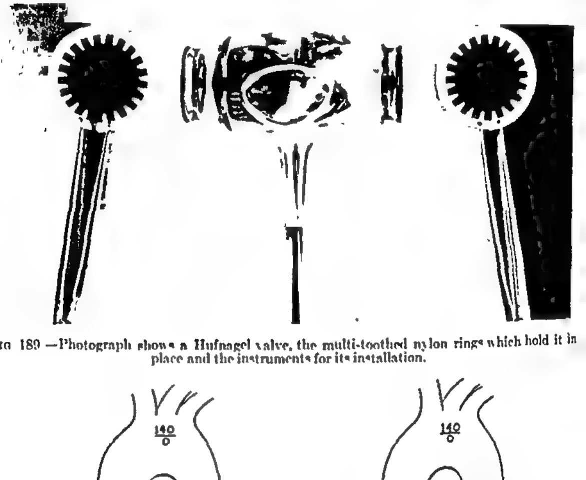

FIG 189 —Photograph shows a Hufnagel valve, the multi-toothed nylon rings which hold it in place and the instruments for its installation.

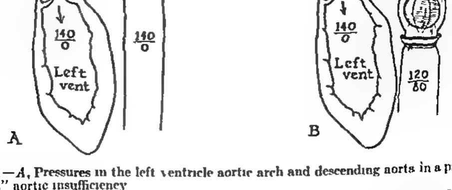

FIG 190 —*A*, Pressures in the left ventricle aortic arch and descending aorta in a patient with "wide open" aortic insufficiency

B, Immediately after the installation of a Hufnagel valve normal pressures are present distal to the valve. *Pressures proximally are unchanged.*

The upper end of the valve is inserted and the nylon retaining ring is put in place. The remainder of the aortic wall is divided and the distal end of the valve is inserted and held in place by the multi-toothed nylon ring. A fine spinal puncture needle which has been bent into a curve is passed through the aortic wall up into the valve; and with a syringe, air is aspirated from the valve and is replaced by normal saline solution. The distal clamp is opened first. The proximal clamp is then removed.

The mediastinal pleura is closed over the valve. The chest is drained by means of an appropriate intercostal tube and the wound is closed in layers.

The complications are those associated with the valve which include infection in the wound, dislodgement of or clotting in the valve, and those associated with the heart disease *per se*. Most patients become accustomed to the clicking due to the motion of the ball in the valve. A coating placed on the ball in the new models of the valve has made them silent. Figure 190 shows the blood pressure readings in the arms and legs before and after the insertion of a Hufnagel valve in a 24-year-old man. His exercise tolerance has increased, he has discontinued the use of digitalis and his heart size and his diastolic murmur have decreased.

QUESTIONNAIRE

1. What are the etiologic factors in aortic stenosis? Aortic regurgitation?
2. What types of deformity may be present in the stenotic aortic valve?
3. What is the normal size of the aortic valve? How small must the orifice be to [illegible]
4. [illegible] aortic stenosis and what is the usual [illegible]
5. Discuss the surgical treatment of aortic stenosis.
6. How is the diagnosis of aortic regurgitation made? What manifestations of it may be demonstrated in peripheral vessels?
7. Discuss the surgical treatment of aortic regurgitation.
8. What does the Hufnagel valve do and what is the basis for improvement after its use?

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CHAPTER 30

SURGERY OF THE HEART AND GREAT VESSELS—TETRALOGY OF FALLOT

By John L. Keeley

This congenital cardiac abnormality was described more than a century ago. It is considered to be the most common cause of cyanotic heart disease in patients who live beyond infancy. The original description included the following four main features: pulmonary stenosis or atresia, dextroposition (overriding) of the aorta, right ventricular hypertrophy and ventricular septal defect.

More recent studies have considered the good possibility that the right ventricular hypertrophy is acquired in the sense that it is a response to obstruction of the right ventricular outflow tract, due to either valvular or infundibular stenosis. Continuing anatomic studies suggest that it is the size and location of the high ventricular septal defect that places both ventricles in communication with the aorta, rather than an anatomic aortic straddling of the ventricular septal defect, the so-called "overriding."

Angiocardiography and cardiac catheterization techniques have increased the accuracy of diagnosis in heart diseases, particularly those of congenital origin. These studies have focused more attention on the altered dynamics of heart disease

The basic disturbance in a typical patient with tetralogy of Fallot may be summarized as follows: Pressure in the right ventricle rises to overcome the resistance of the stenotic pulmonary valve or the narrowing of the infundibular region. The right ventricle becomes hypertrophied and the pressure within it approaches or exceeds that of the left ventricle so that a right to left shunt develops. Unaerated blood from the right ventricle is propelled through the ventricular septal defect and reaches the aorta with ærated blood from the left ventricle (Fig. 191). As stated above, because of the location of the septal defect, blood from the right ventricle may be propelled directly into the aorta through the septal defect. The size of this shunt and the amount of blood passing from right to left determine the degree of peripheral cyanosis.

Recent physiologic studies have shown that patients with tetralogy of Fallot and indeed many with other congenital cardiac defects do not present a constant clinical picture. Nor are the findings constant in any one patient. For example, if the obstruction to the outflow tract in the tetralogy is mild, cyanosis may be absent as blood from the right ventricle is expelled through the pulmonary artery along with blood from the higher pressured left ventricle coming through the ventricular septal defect. This results in an increased amount of blood flow through the lungs leading eventually to pulmonary vascular sclerosis and pulmonary hypertension. This increased resistance is followed by right ventricular hypertrophy and a right to left shunt. Cyanosis then appears.

In some patients the appearance of cyanosis may be delayed until the patent ductus arteriosus closes (Fig. 192). This course of events suggests a less severe degree of stenosis of the outflow tract.

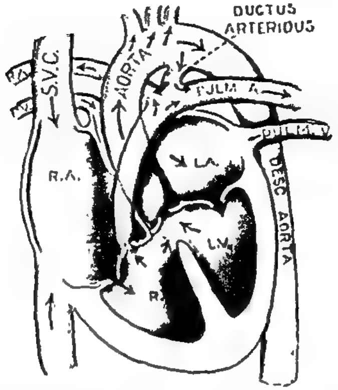

FIG. 191.—The four features of the tetralogy of Fallot are shown: (a) pulmonary stenosis, (b) dextroposition of the aorta, (c) ventricular septal defect and (d) right ventricular hypertrophy. Oxygenated blood reaches the pulmonary artery through the ductus arteriosus and may prevent cyanosis in the early months of life.

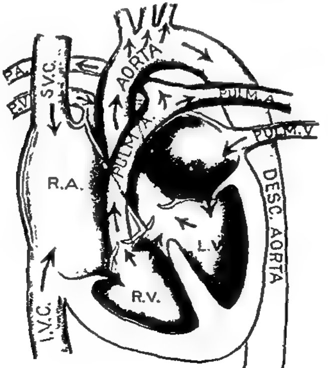

FIG. 192.—The closure of the ductus arteriosus is often followed by the appearance or increase of cyanosis This is due to the greater proportion of unoxygenated blood which reaches the systemic circulation

Infants with tetralogy of Fallot usually show cyanosis of varying degree from birth. Their cyanosis may be increased by feeding, crying, defecation and exertion. They have a systolic murmur and may have episodes of unconsciousness and convulsions, presumably due to cerebral hypoxia. Children develop the squatting habit which serves to compress blood out of the legs and lower abdomen causing it to circulate in central areas to improve its oxygenation. The increased peripheral resistance due to the squatting position may alter the intracardiac dynamics and decrease the amount of the right to left shunt. Dyspnea on exertion and decreased exercise tolerance are usually present.

Most patients with tetralogy of Fallot are underdeveloped. Cyanosis may be mild or moderate and may appear only in the lips and nail beds. Clubbing of the fingers and toes is usually present. The cardiac impulse is more prominent in the xiphoid area than elsewhere due to right ventricular hypertrophy. A thrill at the lower end of the sternum and along its left border is found in a majority of patients. The cyanosis, which is present from birth, may be mild at first and becomes more severe as right ventricular hypertrophy develops and increases the right to left shunt.

A moderately intense systolic murmur due to the pulmonary stenosis or the narrowing of the outflow tract is heard near the middle or lower part of the left sternal border. Both the thrill and murmur depend upon the volume of blood passing through the shunt.

Laboratory studies indicate secondary polycythemia with hemoglobin values up to 25 gms., erythrocyte counts from 8,000,000 to 10,000,000 and varying degrees of decreased oxygen saturation of the arterial blood. Some values for oxygen saturation of arterial blood as low as 25 per cent instead of the normal of 95 per cent have been recorded. A range from 35 per cent to 75 per cent is common in tetralogy.

A roentgenographic study of the chest discloses a small or normal-sized heart with evidence of right ventricular hypertrophy. The pulmonary arterial segment is less prominent than normal and may even be concave. There is decreased vascularity in the lung fields, and in about one-fourth of patients evidence of a right aortic arch and right descending aorta is seen. The electrocardiographic tracing usually shows right ventricular hypertrophy. Cardiac catheterization is not usually necessary to establish the diagnosis of tetralogy of Fallot. However cardiac catheterization in these patients can demonstrate the right to left shunt, the volume of blood flowing through it, right ventricular hypertension, and the type of stenosis, whether valvular or infundibular, in the outflow tract. Angiocardiography may be helpful in demonstrating the size of the pulmonary artery.

SURGICAL TREATMENT

In 1945 Blalock and Taussig reported the anastomosis of the brachial artery to the left pulmonary artery in an effort to send more blood into the lungs for æration. The results were encouraging, even spectacular in some instances. Potts in 1946 succeeded in making an anastomosis between the descending aorta and the pulmonary artery, essentially a "patent ductus arteriosus." The principal feature of these procedures are shown in Figures 193 to 198. Both the Blalock-Taussig and the Potts procedures fail to correct any of the basic abnormalities of the tetralogy of Fallot but add a new one. The operative mortality in the shunt procedures averages less than 15 per cent in all cases.

Brock has attacked the problem by opening the stenotic outflow tract by closed methods in an attempt to correct one of the abnormalities in the tetralogy instead of adding another. A closed method as described for isolated pulmonic stenosis is used in the valvular type of stenosis. In cases of infundibular stenosis an attempt is made to rongeur away some of the hypertrophied muscle of the outflow tract of the right ventricle. Considerable benefit may result if the stenosis is of the valvular type and an adequate opening has been obtained. However the mortality is greater (15 to 25 per cent), and the results are not as consistently good as in the shunt operation.

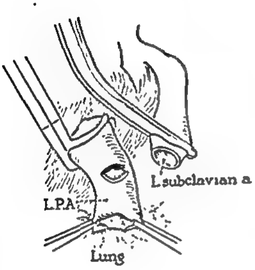

FIG. 103

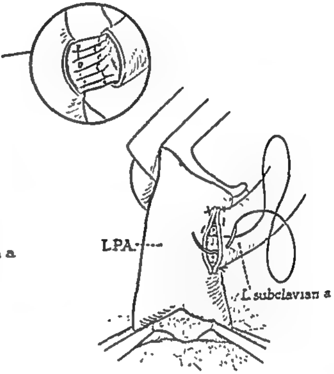

FIG. 104

FIG. 103.—The subclavian-pulmonary artery anastomosis of Blalock as performed on the left side. The subclavian artery has been mobilized and divided as far distally as possible to provide adequate length to reach the left pulmonary artery. Insert shows placement of the posterior suture Approximation is achieved by pulling the suture taut.

Very recently the correction of the abnormalities in tetralogy of Fallot by open cardiotomy has been successfully accomplished by the use of an extracorporeal circulation. The current mortality of about 30 per cent in these attempts will certainly decrease as experience with the method grows. The selection of more favorable cases instead of the desperately ill ones, which have been treated in the majority of instances up to the present time, will also decrease the mortality.

Surgery for tetralogy of Fallot is being withheld in most patients pending the improvement of open methods. The technique of the Blalock-Taussig and the Potts operations are presented here, however, and may still be indicated in specially selected patients under particular circumstances. It is true that the application of these techniques does not offer the prospects of longevity, which correction of the defects would, but one cannot fail to be impressed with the benefit obtained for

patients with tetralogy by these methods of systemic artery-pulmonary artery anastomoses. The reports of series of such patients now followed for approximately ten years attest to the benefit which they have received. Between 80 and 90 per cent of those surviving the operation can lead relatively normal lives. During the present period of change one must pay attention to the mortality rates of all methods in arriving at a decision to employ any of them.

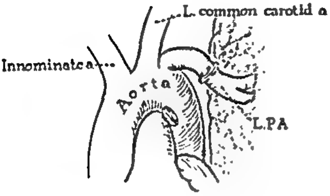

Fig. 195

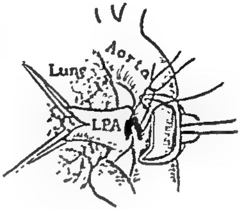

Fig. 196

Fig. 195.—In some instances an end-to-end anastomosis may be necessary because of the caliber or length of the involved vessels. There is danger that such a shunt may be too large and lead to cardiac failure.

Fig. 196.—The aortic-pulmonary artery anastomosis of Potts. If this communication is made with the vessels at right angles torsion of the anastomotic site is less when the lung is inflated and returns to its normal position. The Potts clamp is in place, anastomotic openings have been made and the posterior continuous suture is begun. It is a simple over-and-over stitch which inverts the posterior edges.

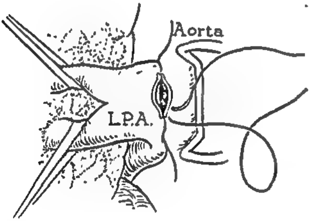

Fig. 197

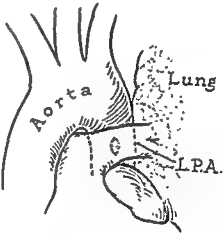

Fig. 198

Fig. 197.—The anterior half of the anastomosis is completed by a continuous running mattress suture of fine silk.

Fig. 198 —The completed aortic-pulmonary artery anastomosis. The stoma represented by the shaded area should not be more than 3 mm. in diameter.

One of the first technical considerations in the performance of systemic artery-pulmonary artery connections is the anatomy which determines the feasibility of these procedures. In general it may be said that the left-sided approach can be used in almost all patients. If the aorta descends on the left as it does in three-fourths of the patietns with tetralogy of Fallot, a Potts procedure can be accomplished (Figs. 196 to 198). If the patient has a right-sided aorta a Blalock-Taussig

procedure can be done (Figs. 193 to 195). In either event, care must be exercised so that the communication is not too large. If it is, heart failure may follow. It may develop rather rapidly with an excessively large shunt. Potts has recommended that an opening 3 mm. in diameter in the aortic-pulmonary artery anastomosis. The exact size of the subclavian-pulmonary artery anastomosis in the Blalock-Taussig procedure is somewhat less important due to the diminished amount of blood which will flow through the subclavian artery compared to that which passes down the aorta.

The Brock procedure of correcting stenosis of the outflow tract of the right ventricle in patients with tetralogy of Fallot is the same as that used in isolated or "pure" type of pulmonic stenosis.

The general availability of pump oxygenators for extracorporeal circulation permitting open cardiotomy will probably make more change in the surgical management of tetralogy of Fallot than in most of the other lesions which have in the past been treated by closed methods.

QUESTIONNAIRE

1. What are the four characteristics of the tetralogy of Fallot? Which of these can be considered acquired?
2. What is the usual history in a patient with tetralogy of Fallot?
3. Describe the physical findings which are commonly present.
4. What can be learned from the x-ray study of the chest, blood gas determinations, and angiocardiography?
5. Is cardiac catheterization necessary for the diagnosis? What information will it provide?
6. What is the basis for the Blalock-Taussig or the Potts operations for tetralogy of Fallot? What objections are made to these procedures?
7. What is the Brock operation in the treatment of tetralogy of Fallot? What advantages does it have over the shunt procedures?
8. What is the ideal surgical treatment for tetralogy of Fallot?

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CHAPTER 31

SURGERY OF THE HEART AND GREAT VESSELS—AURICULAR AND VENTRICULAR SEPTAL DEFECTS

By John L. Keeley

Defects in the auricular and ventricular septa are congenital in origin in practically all instances. The exceptions are rupture of the interventricular septum secondary to infarction and the rare defect due to trauma.

The embryonic atrium is divided into a right and left chamber by the membranous septum primum which grows caudally from the roof of the atrium. The lower part of the septum primum has a curved, crescent-shaped edge which forms the upper border of a defect called the ostium primum. This opening normally closes by further downgrowth of the septum primum and its fusion with the endocardial cushions, which separate the atrioventricular canals into right and left channels.

After the septum primum has made its appearance an opening develops in its upper part. Later a second membrane called the septum secundum develops parallel to, and fuses with the septum primum. This veil of tissue normally closes the ostium secundum. Failure to do so results in an atrial septal defect of the ostium secundum type. This defect is usually centrally placed in the septum and occupies a position similar to that of the normally located foramen ovale.

The average size of an ostium secundum defect is about 3 cm. in diameter but the range may be from 1 cm. in diameter to almost complete absence of the septum. The defect may consist of one large or several small openings. The ostium secundum type of defect is commonly associated with anomalous pulmonary venous drainage.

Should there be failure of the ostium primum to close, a defect will persist in the lower part of the interatrial plane. The mitral and tricuspid valves form the base of this defect.

A third type of abnormality is the most severe type and is due to an early arrest in the development of the heart. The auricular and the ventricular septa fail to fuse so that free communication between the right and left sides of the heart results. If in addition to this the mitral and tricuspid valves fail to fuse, free communication between all four chambers of the heart is present, a persistent atrioventricularis communis.

The flow of blood through a simple atrial septal defect is from left to right due to the higher pressure in the left atrium. This in turn is a reflection of the difference in pressure between the two ventricles. As a result of the left to right shunt, increased flow through the lungs may lead to pulmonary hypertension.

Some children with atrial septal defects have no symptoms but decreased exercise tolerance is a fairly common complaint. In others the development of congestive heart failure and repeated respiratory infections may be the predominant features.

Children with atrial septal defects are usually thin and underdeveloped. The blood pressure is normal but the pulse pressure is narrow. A systolic murmur is

present in both the ostium primum and the ostium secundum types of defect. The murmur of the ostium secundum defect is blowing in character and is of moderate intensity and heard in the second or third interspaces to the left of the sternum. The murmur of the ostium primum defect is harsh and loud due perhaps to mitral regurgitation which is commonly associated with it. It is heard best at the apex and is transmitted to the axilla and sometimes to the back.

Roentgenograms show cardiac enlargement due principally to the size of the right atrium and right ventricle. The enlargement reflects the magnitude of the shunt. There may be some prominence of the pulmonary artery and its branches.

Cardiac catheterization shows increased oxygen content of the right atrial blood. In patients with ostium primum defects and in older patients with either type, pulmonary hypertension may be present.

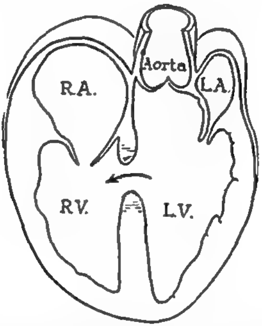

FIG. 199.—Diagram shows the ventricular septal defect in the membranous portion of the septum, the usual site of these abnormalities. Arrow indicates the direction of the shunt prior to the onset of pulmonary and right ventricular hypertension.

Defects in the interventricular septum may occur as isolated abnormalities. They are due to the failure of the interventricular foramen to close. The major portion of the interventricular septum is muscle; but the upper, where defects usually occur, is membranous. A small opening in the membranous portion of the interventricular septum without any other abnormality is called the *maladie de Roger* (Roger's Disease). It is asymptomatic and is compatible with a long and active life.

Patients with larger interventricular septal defects (Fig. 199) usually have pulmonary hypertension and are sick. Underdevelopment, bouts of congestive failure and repeated respiratory infections begin in infancy.

These patients show great cardiac activity. There is a sytolic thrill and a long harsh systolic murmur along the lower part of the left sternal border. Pulmonary hypertension is indicated by the accentuation of the second pulmonic sound and in many patients by hemoptysis.

The x-ray study of the chest shows considerable cardiac enlargement due to the size of the left auricle and both ventricles. Engorgement of the pulmonary vessels

is seen. The electrocardiogram usually discloses right ventricular hypertrophy and in some there is also evidence of left ventricular hypertrophy. Right bundle branch block is not uncommon. Cardiac catheterization discloses a right ventricular hypertension and usually a left to right shunt. The flow may ultimately become reversed as pulmonary resistance increases.

SURGICAL TREATMENT

The obvious surgical procedure of choice is closure of septal defects. A number of highly ingenious methods have been devised for the closure of the ostium secundum anomalies. A variety of closed methods has been presented since surgical attack upon the defect was first suggested by Murray. The names of Bailey, Gross, Swan, Brock and Bjork are associated with the closed techniques which have been used up to the present time.

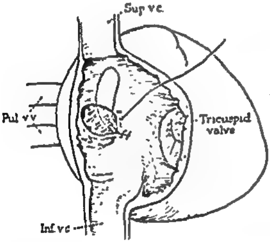

Fig. 200

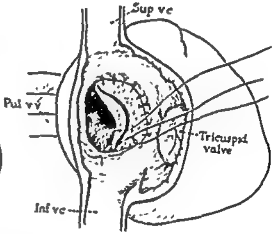

Fig. 201

Fig. 200.—Ostium secundum type of defect can be closed by direct suture during open cardiotomy. The pulmonary veins empty into the left auricle in a normal fashion in this diagram.

Fig. 201.—Wh [illegible]

Closure of an interventricular septal defect has been attempted by Murray and Kay by closed methods with some success.

By means of hypothermia Swan and Lewis have used an open method for atrial septal defects. This provides a period of six to eight minutes during which time the flow of blood returning to the heart can be interrupted, the auricle opened, the defect sutured, and the auricle closed again. The chief objection to this method is the limited time for which the circulation may be interrupted without causing serious brain damage.

The pioneer work of Gibbon in developing a pump oxygenator has formed the basis for the successful use of such apparatus by Lillehei, Kirklin, Dennis and others. Thus open cardiotomy is possible and both atrial and ventricular septal defects have been closed with this method. Figure 200 shows the closure by suture of an ostium secundum defect. Figure 201 shows the placement of an Ivalon prosthesis in a septal defect which was too large for simple closure by sutures.

It has been demonstrated that the repair of intracardiac defects at open cardiotomy and with interruption of the circulation can be done with reasonable risk. The repair of the simple septal defects has already been followed by attack on the more complicated abnormalities.

QUESTIONNAIRE

1. What is an ostium primum defect and to what is it due?
2. Describe the formation of an ostium secundum defect.
3. Describe the history and physical findings in a patient with (*a*) an ostium primum defect, (*b*) an ostium secundum defect, and (*c*) a large ventricular septal defect.
4. What is the *maladie de Roger?* How is it diagnosed and what is the prognosis?
5. What is the surgical procedure of choice in the repair of septal defects and how can these procedures be accomplished?

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CHAPTER 32

SURGERY OF THE CHEST—PHYSIOLOGICAL PRINCIPLES

By George W. Holmes

In addition to the general surgical principles of antisepsis, hemostasis, tissue handling and blood replacement, there are certain basic principles in thoracic surgery which must be thoroughly understood in order to avoid many pitfalls during the care of the injured thorax. In brief, these are: expansion of the lungs, clear airways, unimpaired diaphragmatic function, a rigid chest wall and the desirability of early ambulation.

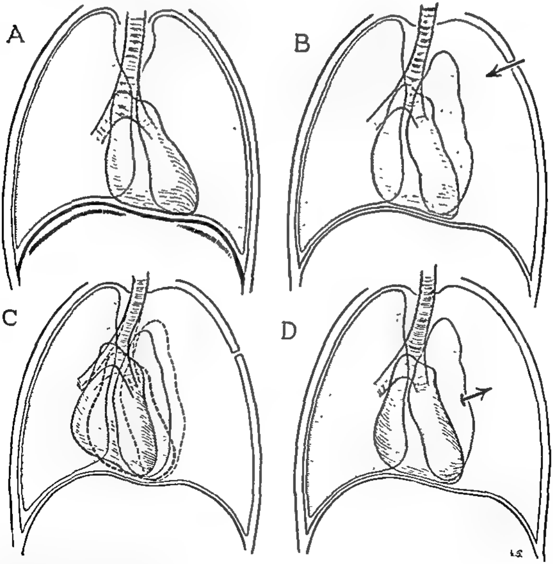

Fig. 202 —Changes in
and shift of mediastinum
of mediastinum associate
sociated with tear of visc

"Physiological Position of Rest" of the Lungs.—A thorough understanding of the function of respiration is absolutely necessary in order to prevent or counteract disturbances which may develop as a result of opening the thoracic cage. In thoracic surgery, it is imperative that the function of the lungs is maintained within normal limits. Normal respiratory function is attained when the lungs are fully expanded within an intact chest cage, the tracheobronchial air passages are unobstructed and the diaphragms are functioning normally. We have termed this state of the lungs as the "physiological position of rest." This is the requirement essential, for instance, when determining the metabolic rate in a normal individual with a normal pulse, blood pressure and respiratory rate. In the "position of rest"

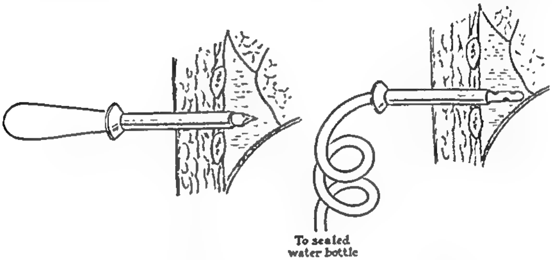

FIG. 203 FIG. 204

FIG. 203.—Water seal drainage. Insertion of trocar.

FIG. 204.—Insertion of catheter through trocar shield.

the lungs can meet any emergency and will continue to do so until pulmonary insufficiency develops. Although voluntary effort can affect the respiration, the most frequent and dangerous factors are those due to metabolic disturbance or adverse reflex stimuli. By increasing the depth and rate of inspiration the lungs can assist in overcoming these metabolic disturbances; more "air" is obtained and the excretion of pulmonary waste products, such as carbon dioxide, is increased.

Pneumothorax, Hydrothorax and Hemothorax.—Fully expanded lungs are necessary for adequate alveolar oxygen and carbon dioxide exchange which occurs between the blood in the pulmonary capillaries and the air in the alveolar portion of the lungs. This is a delicate mechanism whereby, during inspiration, the oxygen is absorbed by the blood in the lungs and transported to tissue cells and carbon dioxide from the blood is eliminated by expiration. If there is interference in this mechanism, such as a partially collapsed lung due to pneumothorax, hydrothorax, or hemothorax, the "position of rest" is impaired and the lungs are unable to cope with the emergency thus developed. Therefore, it is essential for the surgeon to overcome this difficulty by instituting measures which will re-expand the lung fully to the chest wall.

The surgeon must determine if the pneumothorax is due to air entering the pleural cavity either as a result of a penetrating wound of the chest wall (open pneumothorax) or as a result of rupture of the peripheral pulmonary alveoli. When

the intrapleural air does not communicate with the atmosphere, or when the external wound has been occluded the pneumothorax is said to be closed.

The primary consideration in the treatment of pneumothorax is to aspirate the air in the chest cavity as completely as possible. Sucking wounds require immediate closure with skin clips or silk sutures or if this is not feasible, the wound is packed with Vaseline dressings. In case of bronchopleural fistulæ, due to spontaneous rupture of a peripheral bleb or alveoli, manual suction of the pneumothorax

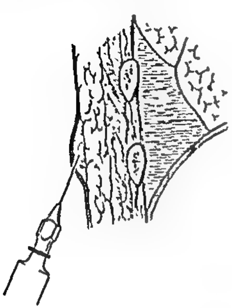

FIG. 205

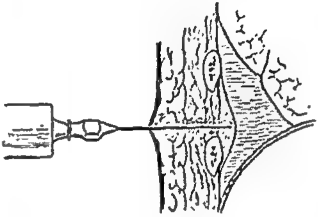

FIG. 206

FIG. 205.—Water seal drainage. Application of local anesthetic to the skin.
FIG. 206.—The anesthesia is carried to the parietal pleura.

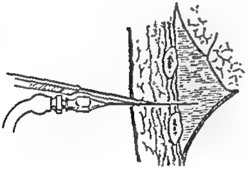

FIG. 207

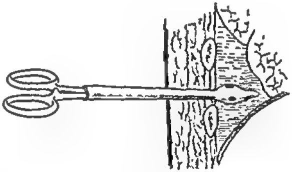

FIG. 208

FIG 207.—A stab wound is made with scalpel by following the anesthetic needle.
FIG. 208.—Introduction of de Pezzer catheter through stab wound.

with a needle and syringe may be too slow or ineffective to expand the lungs. In these cases, a closed water seal should be instituted. This may be accomplished by inserting a mushroom catheter, Foley catheter or a urethral catheter into the chest wall through a small stab incision, through a trocar opening or through the bed of a resected segment of rib. The catheter is secured in place by a silk or wire suture to the skin. The end of the tube is then attached to a sterile tubing which extends under sterile water in a sterile bottle placed on the floor. It is observed

that the water in the glass tubing rises on inspiration and falls during expiration. Upon forced expiration or coughing, bubbles are seen coming from the submerged tube. These bubbles represent pneumothorax air which is being forced out of the pleural cavity. Bubbling may continue for days or until the bronchopleural fistula is healed and the chest cavity is completely free of air. When auscultation and chest x-ray confirms full expansion of the lungs, the catheter is removed. When

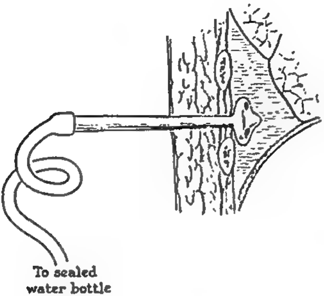

Fig. 209.—de Pezzer catheter in place and attached to sealed water bottle.

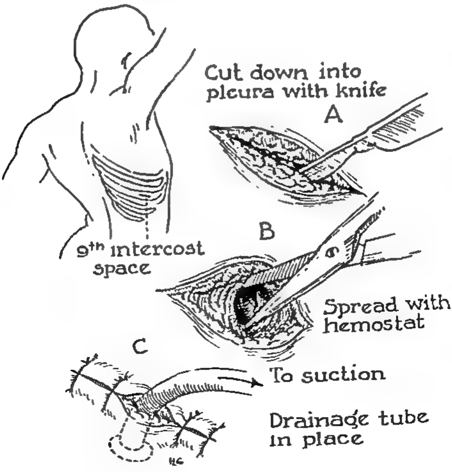

Fig 210 —Closed method of drainage.

removing the catheter, it is important to carefully close the catheter wound to avoid recurrence of the pneumothorax.

At times it may become necessary to insert two or more catheters in the chest to obtain adequate decompression of the pneumothorax and to re-expand the collapsed lung. Pneumothorax resulting from multiple bronchopleural fistulæ may be so extensive that the largest catheter (size 36) is inadequate to accomplish decompression. In this instance, *two or more catheters may be inserted*. Obviously, bilateral pneumothorax requires bilateral closed water seal drainage. A limited

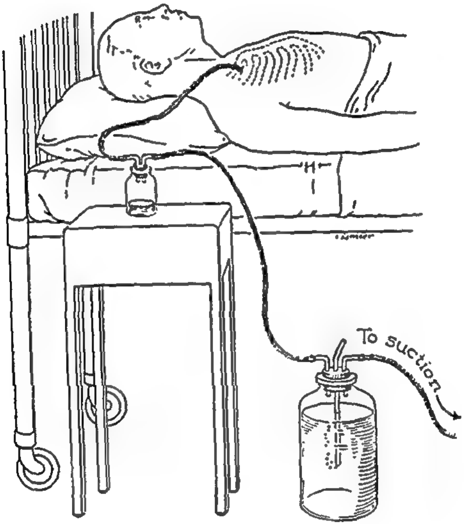

Fig. 211.—Water seal drainage with mechanical suction.

pneumothorax with 25 per cent or less of lung collapsed is not a serious handicap and can be treated by aspiration with the use of a needle and syringe; if it increases, water seal drainage is instituted. Fluid and blood in the thoracic cavity collapsing the lung is removed by multiple needle and syringe aspirations. Water seal drainage with a catheter is rarely successful as fibrin or blood clots obstruct the catheter within twenty-four to forty-eight hours. Closed water seal drainage may introduce infection into the hydrothorax or hemothorax and is not encouraged except in infants.

Surgical Emphysema.—In a closed pneumothorax due to bronchopleural fistulæ, the intrapleural pressure increases causing the mediastinum to shift over to the contralateral side and to encroach upon the opposite lung. The tension pneumo-

thorax thus developed will seek a way out of the chest cavity through the diaphragm into the abdomen causing a pneumoperitoneum or through a defect in the mediastinum causing a mediastinal emphysema. The escape of air may be along the fascial planes of the body leading to a generalized surgical emphysema.

A surgical emphysema may be found along the thoracic fascial planes as a result of internal compound fracture of the ribs. Emphysema is a serious complication; the patient's eyes may be closed tightly by the accumulation of air in the loose alveolar tissue around the eyes and his physical appearance may be completely altered, thus terrifying the patient's relatives.

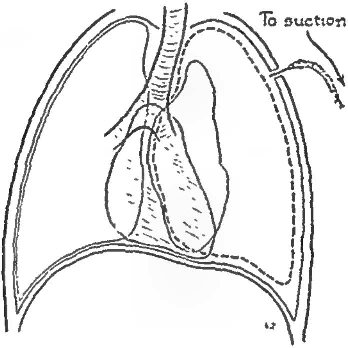

FIG 212.—Relief of traumatic pneumothorax by withdrawal of air through a needle.

Surgical emphysema of the neck and mediastinum impairs the venous return from the head and upper extremities. This may lead to stasis anoxia of the brain and extremities. Deglutition may also be impaired with the result that the patient fails to maintain adequate nutritional and fluid balance. Mental confusion and unconsciousness soon appears if the emphysematous state increases and the tension pneumothorax is not relieved. The shifting of the mediastinum and the increased tension pneumothorax will soon encroach upon and interfere with the normal blood flow of the superior and inferior vena cava and thus decrease the cardiac filling and output. Surgical emphysema requires early and adequate water seal drainage. Multiple superficial incisions of the neck to allow escape of the subcutaneous air or insertion of a rubber Penrose drain into the upper mediastinum along the trachea through supraclavicular incisions have been tried with minimal success. Water seal drainage for the tension pneumothorax is the most important method of treatment. This is combined with oxygen administration by nasal catheter or nasal mask to increase the absorption of the subcutaneous air.

Paradoxical Motion of the Chest Wall.—The "position of rest" of the lung requires a rigid chest wall. If the continuity of the chest wall is interrupted, the lung with its negative intrapleural pressure of the affected side is exposed and neutral-

ized by atmospheric pressure and its efficiency is handicapped. In an effort to overcome this difficulty, the thoracic cage increases slightly in size on inspiration and in this manner attempts to maintain the negative intrapleural pressure essential for lung expansion. If a considerable portion of the bony framework is injured, as occurs by multiple rib fractures, it will be pushed inward by the weight of atmospheric pressure and the descent of the diaphragm during inspiration. The rest of the thoracic cage will increase in size on inspiration. On expiration, the injured portion of the chest cage will be pushed outward by the powerful ascent of the diaphragm while the rest of the thoracic cage decreases in size. This results in paradoxical motion of the injured portion of the thoracic cage compared to the uninjured side during respiration. It is obvious that a profound impairment of pulmonary function is thus established which must be corrected before the maximum "position of rest" of the lungs can be attained. If an open pneumothorax has occurred at the time of the multiple rib fractures, the open pneumothorax must be converted to a closed pneumothorax and treated as described above. The fractured ribs must be splinted by adhesive strapping or a reinforced corset-jacket. The fractured ribs must be splinted to the intact ribs above and below by wooden tongue blades or thin metal strips so that the injured chest can function as a normal intact solid cage. Posterior intercostal nerve blocking at the angle of the rib with 1 per cent procaine may be necessary to relieve pain before attempting to apply tight adhesive strapping.

The basic surgical principles in the treatment of bilateral rib fractures is similar to the unilateral fractures but requires more ingenuity in order to obtain rigidity of the chest wall. In some cases external traction on the chest cage may be necessary. This may be done by grasping the ribs with towel clips and suspending these with rubber bands or springs anchored to an overlying framework. In this manner the chest wall is held in a constant position of inspiration thus overcoming the paradoxical motion. Fractured ribs heal rapidly and in about ten to fourteen days paradoxical motion is usually corrected.

Role of the Diaphragm.—The flow of air to and from the lungs depends entirely upon the rigid thoracic cage and the lungs play a relatively passive role. Air is forced into or out of the lungs in accordance with the pressure differences between the atmosphere and the negative intrapleural space and by the action of the diaphragm and the slight increase in size of the thoracic cage. The diaphragm descends during inspiration, causing an increase in the volume of the thoracic cage and an increase in the negative pleural pressure and as a result more air rushes into the tracheobronchial tree for the inspiratory expansion of the lungs.

The act of expiration is to a large extent a passive movement; the muscles of inspiration of the thoracic cage relax thus, permitting the thorax to seek its own level; the lungs are squeezed down by this and the relaxed diaphragm being pushed upward by the abdominal contents. In forced expiration, the diaphragm is pushed upward into the chest cavity by the strong contraction of the abdominal muscles pushing the abdominal contents up against the diaphragm. The phrenic nerve innervation of the diaphragm must be protected at all times. Loss of diaphragmatic function as a result of injury of the phrenic nerve is rare except that which occurs either as a surgical procedure or as a surgical accident.

The efficiency of diaphragmatic function is interfered with in varying degrees in any of the following conditions: hemothorax, hydrothorax, pneumothorax, fractured ribs, severed phrenic nerve with paralysis of the diaphragm, and trau-

matic rupture of the diaphragm. Obviously, in case of either hemothorax or hydrothorax, the restoration of diaphragmatic function cannot be accomplished until the thoracic cage is freed of fluid by repeated aspirations. Likewise, pneumothorax demands closed water seal decompression. Rigid stabilization of the fractured ribs, if this is a complication, to prevent the paradoxical motion is imperative for good diaphragmatic function. Nothing can be done for paralysis of the diaphragm due to a severed phrenic nerve. However, diaphragmatic motion can be preserved by maintaining the chest wall intact and the lung expanded until regeneration of the injured phrenic nerve can take place; this is especially true in unilateral paralysis. As a rule no serious complications result from this latter condition, except intermittent accumulation of lower bronchial secretion which may require bronchial aspiration. Bilateral diaphragmatic paralysis may be incompatible with life or at best only with limited activity.

TRACHEOBRONCHIAL OBSTRUCTION

It is obvious that the "position of rest" of the lungs cannot be maintained unless the tracheobronchial air passage from the larynx to the alveoli is clear. The most common cause of obstruction is from mucus plugs which may occur anywhere along the air passages. Regurgitation of food or gastric juices, and aspiration of foreign bodies as may occur in general surgery, can also occur in thoracic surgery. Mucus plugs or inspissated purulent secretion is more of a problem in surgery of suppurative pulmonary disease. During surgery, hypercarbia, hypoxia or anoxia may manifest the tracheobronchial obstruction and the observant anesthesiologist corrects the condition by tracheobronchial aspiration.

Chest pain such as that due to broken ribs which causes splinting of the chest muscles and inhibition of the cough reflex, should be relieved by procaine (1 per cent —10 cc.) intercostal nerve block and coughing encouraged. Naso-pharyngeal-laryngeal stimulation with a catheter will encourage a weak or inhibited cough reflex. Inhalation of oxygen-carbon dioxide gas mixture to increase respiration often stimulates a cough reflex.

Bronchoscopic aspiration of the lower air passages with or without local anesthesia is usually deferred until all of the above methods have been tried to effectively cough out all retained secretions in the lower air passages.

Constant intermittent expectoration of mucus or purulent secretion should be insisted upon because retained secretions in the lower air passages for twenty-four to forty-eight hours is followed by varying amounts of pneumonitis. Oral expectorants and inhalation of detergent vapor help to liquify the thick secretions.

Tracheotomy is indicated only in the injured unconscious patient or the patient too weak by debility or neuromusculopathy to cough. Routine tracheotomy in all major postoperative cases is not indicated. The tracheotomy patient is dependent on intermittent manual aspiration of the lower air passages throughout the day and night. The cough reflex is reduced to zero because effective tracheobronchial pressure can not be built up for the sudden explosive effect of the cough. Small tracheotomy tubes are used to avoid or minimize this loss of effective cough reflex, but then respiratory obstruction through or around the smaller tracheotomy tube may also become a problem. Crusting of the tracheal mucosa with increased mucus secretion aggravates the problem of maintaining clear air passages in tracheotomized patients. In the unconscious patient due to brain damage or

drug overdosage, or in the patient with neuro-muscular disease and weakness so that efforts at coughing are ineffective, tracheotomy is done if a daily bronchoscopy, or more, is necessary to maintain clear air passages.

While the air passages are being kept clear by any or all of the above procedures, alveolar bronchiole obstruction may result from spasm and lead to as much obstruction as retained secretions. Broncho-dilatory drugs must be used and the wheezing and dyspnea are soon relieved, but these drugs or other medications are not substitutes for thorough mechanical aspiration of the air passages. Opiates and other drugs that depress the cough reflex or tracheobronchial secretions must be given with extreme care as the retained mucus plugs lead to sub-clinical atelectasis and then clinical pneumonitis.

In thoracic surgery, the surgeon attempts to keep the lung at the "position of rest." During surgery, the lung is partially expanded all the time and periodically fully expanded. Following injury or surgery, the surgeon attempts to leave the lung the same as it existed prior to injury or surgery. It is essential in severe thoracic injury that the surgeon restore the lung back to the physiological position of rest as soon as possible as the patient may have to undergo general anesthesia for abdominal exploration or reduction of fractures.

QUESTIONNAIRE

1. What is meant by "physiological position of rest"?
2. When is normal respiratory function attained?
3. Discuss the importance of normal respiratory mechanism?
4. How may pneumothorax develop?
5. What is the primary objective in the treatment of pneumothorax?
6. How is a sucking wound of the chest treated?
7. How would you treat a bronchopleural fistula?
8. How would you treat pneumothorax resulting from multiple bronchopleural fistulæ?
9. What is tension pneumothorax?
10. How is it developed?
11. What is the etiology of mediastinal emphysema?
12. What are the signs and symptoms of emphysema?
13. What are the possible effects of emphysema?
14. What is the treatment for the above?
15. Define "paradoxial motion of the chest wall"?.
16. What is the etiology and physiology of the above?
17. Give the treatment for the above.
18. Discuss the role of the diaphragm during respiration.
19. How is the diaphragmatic function interfered with?
20. What are the common causes of tracheobronchial obstruction?
21. Discuss the general principles in the treatment of the above condition.
22. When is tracheotomy indicated?

CHAPTER 33

SURGERY OF THE CHEST—SURGICAL ANATOMY

By George W. Holmes

The knowledge of human anatomy is so vital for the understanding of all pathological disorders or conditions where surgery might be helpful. To the surgeon, the knowledge of anatomy is a necessity and a constant challenge to master the details. Only some of the essential thoracic anatomy for the surgeon will be described as many excellent anatomical textbooks and anatomical atlases are available.

The scapula covers the posterior and upper half of the thoracic cage. It is attached posterior to the spine by the trapezius, rhomboid, levator scapula and fibers of the latissimus dorsi muscles, thereby impeding the surgical approach to the underlying ribs. Otherwise all other ribs are quite accessible surgically. Normally, the ribs and interspaces vary in size but deformity of the spine or chronic pleuro-pulmonary disease may greatly alter the size of the ribs and interspaces.

Knowledge of the anatomy of the sternum and the thoracic cage is necessary to orient oneself in making the correct thoracotomy incision. The angle of Louis which is the anterior attachment of the 2nd rib to the sternum is a constant elevated landmark and a starting point to identify ribs. Other methods of counting upward from the 12th rib may be confusing. The distance between the internal mammary arteries varies and must be contemplated in any vertical or horizontal sternal splitting incision for exposure of the mediastinum or heart.

The intercostal arteries arise anteriorly from the internal mammary artery and posteriorly from the descending thoracic aorta and anastomose along the intercostal space. Many perforating branches are given off to the peripheral chest wall muscles and internally to the parietal pleura. The intercostal nerve is closely adherent to the artery and vein. All are protected by a shelving portion of the inferior edge of the rib beginning at the posterior angle.

The muscles of the chest wall are important from the standpoint of their thickness and in number in a given area of the chest. Surgical incisions in the lower axillary or the infra-mammary area will encounter a thin muscle layer over the ribs as contrasted to a posterior chest incision. The muscles holding the scapula to the chest should be preserved whenever possible so as to minimize postoperative disability.

When the thoracic cage has been opened the first anatomical structure to identify is the phrenic nerve. It is always anterior to the pulmonary hilum and inferior pulmonary ligament. It extends along the entire mediastinum into the anterior portion of the diaphrgam. In obliterative pleuritis, the surgeon is cautioned of the possibility of the phrenic nerve being adherent to the mediastinal surface of the lung. Every effort should be made to protect the phrenic nerve from surgical injury because of the importance of diaphragmatic motion postoperatively. Pericardial enlargement or cardiac enlargement, may push the phrenic nerve posteriorly

but its relation to the pulmonary hilum and inferior pulmonary ligament remains the same.

The vagus nerves are seen to be posterior and close to the phrenic nerves in the upper mediastinum, but the former pass downward and posterior to the hili and in the mid-thoracic region follow the esophagus downward. The phrenic nerve runs parallel to the sternum and always anterior to the hili.

The azygos vein and its branches are easily seen in the right upper mediastinum after reflection of the thin mediastinal pleura. The azygos vein is approximately half the size of the superior vena cava vein. The azygos-vena caval junction is just superior to the right hilum. The azygos system is thin-walled and divides into a superior and an inferior branch which extend upward and downward on the anterior lateral surface of the dorsal spine. In the mid-thoracic area the hemi-azygos vein crosses over from the left anterior lateral surface of the dorsal spine

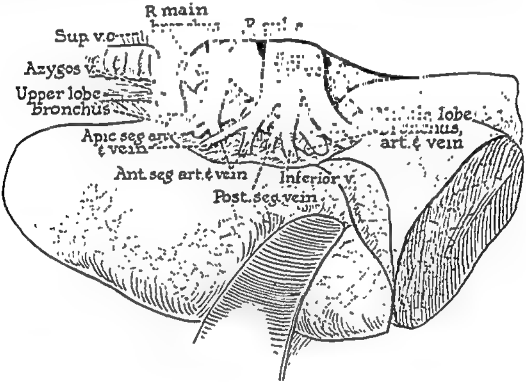

FIG. 213.—Right anterior hilum.

and drains into the azygos system. The latter drains all the blood from the intercostal veins, vertebral veins, and has numerous collateral branches with other venous systems. Bleeding from the azygos system is never forceful but a continuous welling up of blood in the thoracic cavity and associated with early shock.

The aorta is readily seen and palpated through the mediastinal pleura and its arch is just superior to the pulmonary hilum.

In the right chest the junction of the phrenic nerve to the point where the azygos vein enters the superior vena cava determines the upper portion of the right hilum. Atelectasis, apical adhesions, or intrapulmonary cysts may twist or distort the intrahilar structures, but the hilum maintains its relations to the phrenic nerve and azygos system except in the rare condition of an intrapulmonary azygos vein.

In the left chest, the point where the phrenic nerve crosses the aorta determines the upper portion of the left hilum. The hilar structures may be distorted by

pathological pulmonary conditions, but the hilar relations to the aorta and phrenic nerve remain the same.

When the mediastinal pleura is opened posterior to the phrenic nerve in the area of the hilum, branches of the superior pulmonary vein come into view. As the mediastinal pleura is extended posteriorly and inferiorly, the inferior pulmonary vein

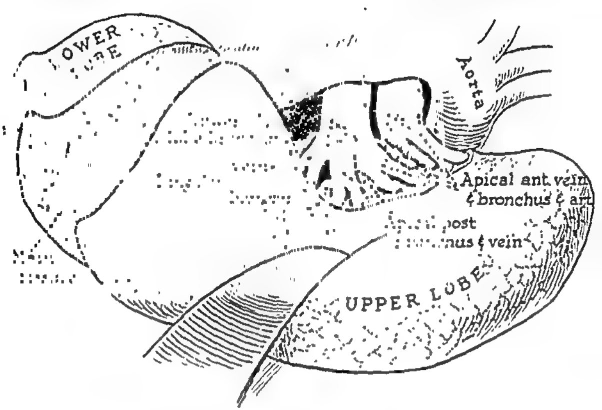

Fig. 214.—Left anterior hilum.

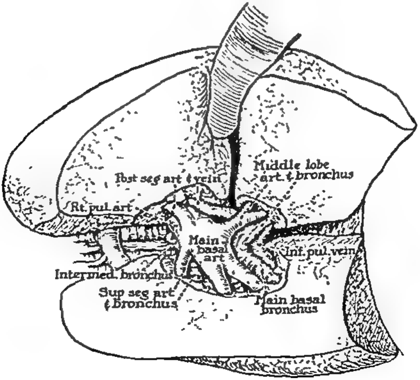

Fig. 215.—Right interlobar hilum.

is seen. From the caudal side of the inferior pulmonary vein, the inferior pulmonary ligament takes its origin and extends to the diaphragm.

A posterior view of the hilum reveals the main bronchus and some of its first branches covered by mediastinal pleura. If this pleura is opened caudally, the inferior pulmonary vein and the inferior pulmonary ligament are encountered.

The anatomical details of the hilar vessels and bronchi are extremely important for pneumonectomy, lobectomy, and segmental lobectomy.

The following comparison may be helpful.

Right Hilum	*Left Hilum*
1. Inferior to azygos vein, is seen the superior pulmonary artery to right upper lobe and adherent to right upper lobe bronchus.	1. Entire left pulmonary artery is somewhat parallel to underside of aortic arch.
2. Superior pulmonary vein completely covers the inferior pulmonary artery branch and requires very careful dissection to separate vessels.	2. Superior pulmonary vein is inferior to the pulmonary artery and easily separated from the artery.
3. Superior pulmonary vein is short and round. This is due to the right upper posterior segmental vein branch. This branch is partially adherent to the underlying artery and makes the separation of the vessels difficult.	3. Superior pulmonary vein is longer, flat and not as wide. It is partially adherent to the underlying bronchus but is easily dissected free.
4. Superior pulmonary vein is wide due to the separate right middle lobe venous drainage. This branch may be separate from the right upper lobe branch into the pericardial sac and is best ligated separately from the right upper lobe venous branch.	4. Superior pulmonary vein can be dissected in one segment as the lingular branch is separate but does not extend as a separate branch into the pericardial sac. The entire vein can usually be ligated with a single ligature.
5. The lobar and segmental lobar veins are easily identified. Anomalies in number of veins or drainage of veins may occur.	5. The lobar and segmental lobar veins are easily identified. Anomalies in number of veins or drainage of veins may occur.
6. The pulmonary artery has two separate branches. The superior branch supplies the right upper lobe (apical and anterior segments). The inferior branch supplies the right upper lobe (posterior segment) right middle lobe and right lower lobe.	6. The pulmonary artery is a single vessel with branches coming off at intervals to the left upper lobe and left lower lobe.
7 The pulmonary artery is ligated by ligatures around the superior and inferior branches separately for a pneumonectomy. The artery is partially adherent to the underlying right main bronchus and its branches.	7. The pulmonary artery is easily ligated with one ligature for pneumonectomy. The artery is barely adherent to a small area of the superior portion of the left bronchus, superiorly in the same area the recurrent laryngeal nerve must be protected.
8. Lobar and segmental lobar arteries are more closely adherent to the respective bronchi than the veins.	8. Lobar and segmental lobar arteries are more closely adherent to the respective bronchi than the veins.

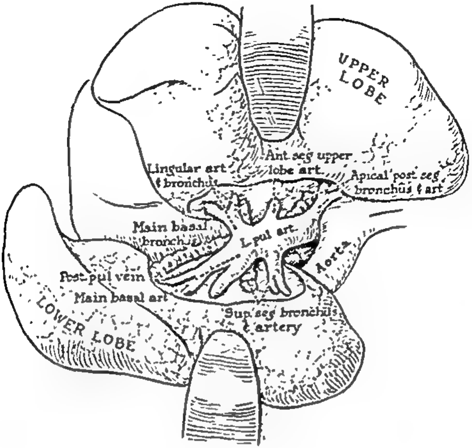

FIG. 216.—Left interlobar hilum.

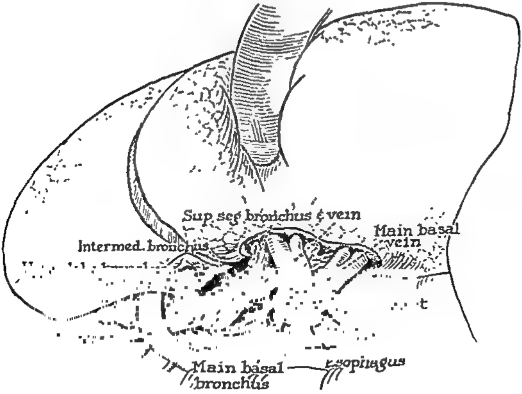

FIG. 217.—Right posterior hilum

The bronchus and its lobar and segmental branches are best identified from the posterior aspect of the hilum but can be identified easily after the respective vein and artery have been ligated and divided.

The Huber-Johnson bronchial classification is followed.

The esophagus and right vagus nerve can be easily mobilized after ligation and division of the azygos vein and incision of the mediastinal pleura posterior to the pulmonary hilum. By anterior displacement of the right lung the entire esophagus can be mobilized and its segmental arterial blood supply arising from the aorta can be identified and ligated.

Dissection of the parietal pleura along the paravertebral area of the chest wall reveals the sympathetic nerve chain with the ganglia and branches.

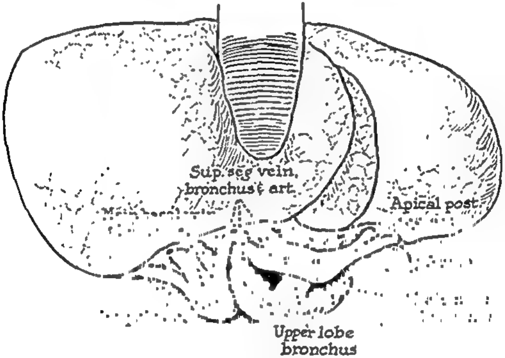

FIG. 218.—Left posterior hilum.

The thoracic duct lies adjacent to the dorsal spine in the posterior mediastinum in a groove between the esophagus and aorta.

Lymph nodes are present in the hilar areas on the medial side of the bronchi. A lymph node is always present at division of the right pulmonary artery into the superior and inferior branches. Smaller lymph nodes are encountered in the hilum of the lung at the beginning of the lobar and segmental lobar bronchial branches. Lymph nodes may extend upward paratracheally or downward along the inferior pulmonary ligaments.

The bronchial arteries are seen closely adherent to the main bronchi near the carina. More distally the bronchial arteries are less discernible except on sharp division of the bronchi and point of bleeding noted.

The diaphragm is composed of muscle which is attached to a strong central tendon. This muscle is rhythmically contracting except posteriorly in the area of the crura. Contracture of the muscle pulls the central tendon down and slightly anterior. The phrenic nerve leaves the mediastinal surface and enters the diaphragmatic muscle anteriorly. Any incision in the central tendon does not impair diaphragmatic motion. Any radial incision through the muscle and tendon of the diaphragm leaves the posterior portion of the diaphragm paralyzed.

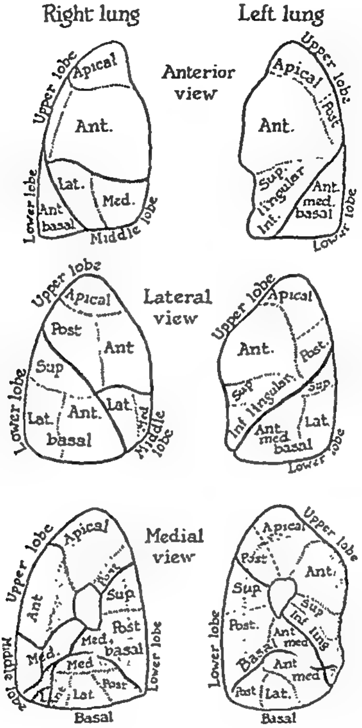

FIG. 219 —Pulmonary segments of the lobes.

QUESTIONNAIRE

1. Give the surgical anatomy of the scapula.
2. What is the angle of Louis?
3. What is its significance?
4. Which part of the chest wall has the least muscle coverage?
5. How would you identify the phrenic nerve?
6. Give the course and relationship of the vagus nerve.
7. How can you identify the azygos vein and its branches?
8. Give the relationship of the aorta.
9. In the right chest, how is the position of the superior vena cava determined? In the left chest?
10. Describe the surgical anatomy of the hilar vessels and bronchi of the right and left hilum.

CHAPTER 34

SURGERY OF THE CHEST—DIAGNOSTIC PROCEDURES

By George W. Holmes and William B. Buckingham

Prior to surgical intervention, it is necessary for the surgeon to know as much as he possibly can learn about the patient and his thoracic disease. Immediate thoracotomy with exploration for diagnosis is often costly and unfruitful; it is discouraged except as the last diagnostic procedure and after all other tests have proved negative in establishing a diagnosis. There are certain specialized diagnostic procedures that will help to establish the diagnosis and extent of involvement of intra-thoracic disease These procedures are as follows:

1. Skin tests
2. Bronchoscopy
3. Esophagoscopy
4. Bronchography
5. Venous pressure and circulation time
6. Angiocardiography
7. Cardiac catheterization
8. Scalene lymph node biopsy
9. Pleural biopsy

In a given patient it is never necessary to perform all of these procedures, but before being subjected to thoracotomy, each patient should have any of these procedures performed that would establish the diagnosis or reveal the extent of the pathology. Exploratory thoracotomy is justified only if the results of the appropriate diagnostic procedures are inconclusive and if neoplastic disease is strongly suspected. However, these procedures should not be substituted for therapeutic measures and used as a means of prolonged procrastination.

SKIN TESTS

Persons who have contracted tuberculosis or any of the fungi diseases demonstrate a lasting skin sensitivity to antigenic preparations of the microörganism. This skin sensitivity begins after the infection and persists beyond the duration of active infection. A positive skin test does not mean active infection with the organism from which the antigen was prepared, nor does it mean that the pulmonary disease under study is caused by this organism, but it does mean past or present contact between the individual and the organism. This is particularly true about the tuberculin test and tuberculosis, but also applies to histoplasmosis, coccidiomycosis and blastomycosis. A negative reaction to these skin antigens in dilution of 1:100 is evidence that there has been no contact between the patient and the organism. These tests are especially important in isolated, circumscribed "coin" lesions, mediastinal lymphadenopathy and localized chronic inflammatory lesions.

mediastinum and into the deep cervical lymphatics and lymph nodes. The scalene lymph node biopsy is carried out to obtain one of the small upper mediastinal glands, if present, for microscopic study.

Technique.—With the patient in position as for a thyroid operation, a 2- or 3-inch incision is made under local anesthesia in the supraclavicular fossa, along the posterior border of the sternocleidomastoid muscle. The clavicular half of the sternocleidomastoid muscle is divided so that the scalene fat pad and the internal jugular vein are visualized. The fat pad overlying the scalenus anticus muscle is separated from the jugular vein. The fat pad is retracted laterally and the jugular vein is gently retracted medially so that its underside can be dissected free. When the jugular vein has been freed on its lateral and posterior sides, the index finger can be passed under the jugular vein into the upper mediastinum so that the lateral wall of the trachea, the carotid and subclavian arteries are palpable. Any lymph nodes encountered are carefully removed by blunt dissection. If no lymph glands are encountered, a portion of the scalene fat pad is excised and sent to the laboratory for microscopic study.

The number of positive microscopic findings has been high and conclusive in cases of lymph nodes resected from under the jugular vein in the upper mediastinum. During the surgical procedure, tedious careful dissection is necessary to prevent troublesome bleeding. Tearing of the parietal pleura can lead to pneumothorax; tearing of the internal jugular vein can lead to hemorrhage and air embolism. Injury to the brachial plexus and its branches and the phrenic nerve must be avoided. On closing the incision, the cut fibers of the sternocleidomastoid muscle are approximated with interrupted silk and the skin edges closed in the same manner.

PLEURAL BIOPSY

Pleural biopsy is more conclusive than the chemical, microscopic and bacteriological studies of pleural fluid.

Technique.—With the patient lying in a lateral position, and under local anesthesia, an incision of 4 to 6 inches is made along the course of the posterior rib overlying the area of pleural fluid or thickened pleura. The chest wall muscles are separated and the selected rib exposed. The intercostal nerves and interspace above and below the selected rib are thoroughly infiltrated with 2 per cent procaine. A segment of the rib, approximately one to two inches, is resected subperiosteally. A size No. 15 aspirating needle is introduced through the subperiosteal bed for a distance of ½ inch to determine the thickness and density of the parietal pleura and also to aspirate any fluid in the thorax. A wedge of the periosteal bed and the parietal pleura are resected for microscopic study. If fluid or pus is encountered, a large de Pezzer catheter is inserted into the chest cavity through the biopsy opening and attached to a water seal system. The chest wall muscle layers are closed around the tube with interrupted silk sutures and the skin edges closed with interrupted silk sutures. The drainage tube is left in the chest for two or three days and then removed in the usual way.

QUESTIONNAIRE

1. What tests may be used to establish the diagnosis of intrathoracic disease?
2. When is an exploratory laparotomy justified?

3. Discuss the value of skin tests.
4. When should bronchoscopy be performed?
5. When should esophagoscopy be performed?
6. When is bronchography indicated?
7. Discuss the value of estimating venous pressure and circulation time.
8. How is angiocardiography performed?
9. How is cardiac catheterization performed?
10. What is the purpose of scalene lymph node biopsy?
11. Describe the technique for the above.
12. What is the value of pleural biopsy?
13. Describe the technique of the above.

CHAPTER 35

SURGERY OF THE CHEST—PRE-OPERATIVE EVALUATION AND OPERATIVE CARE

By George W. Holmes and William B. Buckingham

After the thoracic surgeon has reviewed the chest roentgenograms and completed one or more of the appropriate diagnostic tests, he decides whether the case may be benefitted by major thoracic surgery. The preoperative evaluation may reveal that the patient suffers from other diseases that contraindicate surgery, or which might seriously complicate the surgical and post-surgical management. This preoperative evaluation includes all of the accepted general surgical work-up and in addition:

1. Pulmonary function evaluation
2. Cardiac evaluation
3. Renal evaluation
4. Hepatic and metabolic evaluation

PULMONARY FUNCTION EVALUATION

A careful physical evaluation with particular reference to the chest is helpful in evaluating pulmonary function. Observation of the rate and depth of expiration during voluntary hyperventilation will reveal much about the ventilatory mechanism. This can be supplemented and extended by careful fluoroscopic examination, including examination during voluntary hyperventilation. The amount and character of diaphragmatic motion should be noted, and unequal distribution of inspired air can be detected by unequal "lighting" of various portions of the lung.

When the disease under surgical consideration is localized to one lobe, or less, and the above procedures do not reveal any evidence of diffuse or generalized disease, then it is safe to assume that the patient has sufficient pulmonary function to withstand the contemplated surgery. However, when either the history, physical, *x*-ray, or fluoroscopic examinations reveal evidence of pulmonary emphysema or generalized pneumofibrosis, the clinical estimation of pulmonary function is inadequate and quantitative measurements of pulmonary function must be made.

There are many good tests of pulmonary function. A simple, but adequate measurement of ventilatory function can be made by determining the vital capacity and the maximum breathing capacity. The vital capacity (VC) is the greatest amount of air that can be exhaled in a single breath, following a maximal inspiration. The maximum breathing capacity (MBC) is the total amount of air that can be moved in or out of the lungs in one minute of voluntary hyperventilation. Thus, the VC is a static measurement of volume only and the MBC is a dynamic measurement which greatly depends upon the maximum number of breaths the individual is able to take in one minute.

The normal VC is 3.5 to 4.5 liters, depending upon height and weight of the patient. The normal MBC is 100 to 150 liters per minute, decreasing sharply with age, but also dependent on height and weight. A reduction of VC below 2.5 liters is evidence of severe replacement of lung tissue. If the MBC is above 100 liters per minute, the patient is a good ventilatory risk even for the most extensive resectional procedures. If the MBC is between 50 and 90 liters per minute, the patient must be observed carefully during and after surgery, but is only a slightly greater surgical risk. If the MBC is below 50 liters per minute, major resectional surgery is contraindicated. Procedures, such as decortication, that will improve pulmonary function may be attempted. In the majority of cases MBC and VC will give sufficient information about pulmonary function to guide the surgeon in his decision. In some specific instances more extensive pulmonary function evaluations may be necessary and these may include blood gas determinations, bronchospirometry and test of distribution and diffusion.

CARDIAC EVALUATION

The first question to be answered in the cardiac evaluation is whether or not significant heart disease exists. The heart may be considered normal if:

1. There is no history suggesting coronary artery or other cardiac disease.
2. Physical examination reveals normal rate, rhythm, and tones, and fails to reveal any evidence of heart failure.
3. The EKG is normal or contains only minor nonspecific abnormalities.

If the above conditions are not met, then the type of heart disease and its effect upon the circulation must be determined. Probably the most frequent type of heart disease encountered is coronary artery disease. If the patient has had a myocardial infarction in the part, but has no angina or evidence of heart failure, and has a stable EKG, major resectional surgery may be performed. Severe angina or congestive heart failure must be successfully treated before surgery is performed, and an unstable EKG contraindicates major surgery.

Cor pulmonale is often encountered in association with diffuse pulmonary disease. EKG evidence of right ventricular hypertrophy alone is not sufficient to contraindicate surgery, but it should warn the surgeon to limit intravenous fluids and avoid episodes of anoxia during and after surgery. When the EKG pattern includes right heart strain and/or right bundle branch block, the danger of heart failure is eminent and the patient is a borderline risk. When these EKG patterns are associated with roentgen signs of right heart enlargement or clinical evidence of right heart failure, then major surgery is not indicated.

Surgery in the presence of rheumatic, congenital, luetic, or other types of heart disease will depend upon the over-all effect upon the circulation and this must be evaluated individually.

RENAL EVALUATION

The patient with normal functioning kidneys can withstand the blood transfusions and intravenous infusions that are necessary during the course of major thoracic surgery. This will be too difficult a task for the kidneys of a patient with frank renal failure and uremia. The borderline cases may be best evaluated by observing the results of the Mosenthal and urea clearance tests. If the Mosenthal

test reveals a failure to concentrate above 1.010, and the urea clearance is less than 50 per cent of normal, then surgery is not indicated.

The PSP excretion test gives a good over-all estimation of excretory function It is seldom necessary to perform any of the more elaborate tests of renal function.

LIVER AND METABOLIC EVALUATION

The liver is the center of many metabolic processes, but probably the most important of these is the formation of the plasma proteins. The best screening test of liver function is a determination of the plasma proteins and the albumin and globulin ratio. If this is within normal limits, it is safe to assume that liver function is adequate for surgery. If this is abnormal, then further tests of liver function must be performed.

Diabetes mellitus is the most important metabolic disease that the chest surgeon encounters. A fasting blood sugar and if necessary a glucose tolerance test should be performed on all patients who are to undergo thoracic surgery. If diabetes is present it must be successfully controlled before, during and after surgery in order to lessen the number of infectious complications that occur.

OPERATIVE CARE

Once the surgeon has made the decision to operate preoperative medication is ordered that will allay the patient's anxiety, but not completely suppress the cough reflex or diminish respiration to the point that hypoxia is present. The thoracic surgeon must assist the anesthesiologist until the endotracheal tube has been inserted and a closed circuit anesthesia obtained. Gas anesthesia or intravenous Pentothal with curare-like drugs may be used to relax the patient so that the larynx can be exposed and intubated. In either case, the surgeon must be sure the endotracheal tube is inserted skillfully and quickly without producing a laryngeal spasm, bronchial spasm, or anoxia with cyanosis.

Oximeter tests have shown that the blood oxygenation drops considerably during intubation of the patient. The operative risk is greatly increased even before an incision is made if the patient undergoes a period of anoxia with cyanosis during the endotracheal intubation; it is usually wise to defer the surgery for a few days and re-evaluate the patient.

The positioning of the patient is the surgeon's responsibility and every detail must be observed so that the position is maintained throughout the surgical procedure. After suitable antiseptic skin preparation and draping, the skin incision is made with proper hemostasis.

On opening the pleural cavity, the surgeon must inform the anesthesiologist if there is any arrhythmia of the heart, cyanosis of the blood, shifting of the mediastinum, atelectasis of the lung, overdistention of the lung, or arrhythmia of respiration. Likewise, the anesthesiologist must notify the surgeon if there is any drop in the blood pressure, arrhythmia or impaired air passage flow. It is advisable for the surgeon and the anesthesiologist to both know how much intravenous blood and fluids that the patient has received. If the heart rate slows or becomes irregular, it indicates anoxia; this condition is usually associated with drop in blood pressure. The anesthesiologist must increase the oxygen flow and the concentration, likewise, he must use positive pressure and thereby expand the lung

on the operated side. Intravenous atropine should be given at this same time to increase the heart rate and decrease the chance of sudden neuro-reflex-cardiac stand-still. The surgeon and his assistants should stop all operative manipulations until the heart rate is back to normal or faster and the blood pressure is normal. If the heart stops completely, immediate cardiac massage is started by opening the pericardium through an incision parallel to the phrenic nerve; the heart is gently grasped and milked upward from the apex toward the auricles. The heart is never fully released at any time so as to prevent overdilation and overdistention. Cardiac massage by the milking-contracture of the hand is continued until heart beats are felt and become stronger. The hand is continued in position as a cradle under the heart and not removed until the heart beats are forceful and rapid. Adrenalin (1:1000) in 5 to 10 cc. of saline is injected into the left ventricle as soon as cardiac massage starts and soon thereafter, 10 cc. of 2 per cent procaine is injected into the right and left ventricle. Calcium chloride solution and potassium chloride solution may be injected directly into the left ventricle during prolonged massage. If fibrillation of the heart muscle is noted, electrical defibrillization of the heart is immediately carried out; the fibrillation may stop and further cardiac massage be resumed. If the heart fails to start beating or fibrillating after prolonged massage and shows some dilation, rhythmical electrical shocks by means of the electrical stimulator may be tried followed by further cardiac massage if necessary. During this time, the anesthesiologist maintains positive pressure oxygen into the lungs. This combined treatment for cardiac arrest has been continued for ten hours in a reported case with complete recovery of the patient.

During surgery and following application of the dressing to the wound, the surgeon must be ready to bronchoscope the patient if the anesthesiologist finds that aspiration through the endotracheal tube is ineffective. The danger of a fragment of tumor breaking off in the diseased bronchus and falling into the opposite bronchus to cause obstruction must be considered. The endotracheal tube is left in place until the patient is awake, coughing, and beginning to struggle. Thorough suction of the air passages is done before the endotracheal tube is removed, and respiration observed for any evidence of obstruction. Routine postoperative bronchoscopy is not done and is rarely necessary unless the endotracheal tube is removed before the patient is awake and incompletely "coughed" out.

All water-seal bottle tubes must be clamped before the patient is moved from the operating room to his bed; care must be used during the moving to prevent dislodging or pulling out of a chest catheter. A slight drop in blood pressure occurs during the moving and may require a vascular stimulant (neo-synephrin).

QUESTIONNAIRE

1 What factors must be considered preoperatively in evaluating the patient's condition?
2. How is the pul[illegible]
3. When is it safe [illegible]
4. What is the sig[illegible] in estimating pu[illegible]
5. Give the various tests for pulmonary function.
6 Define: "vital capacity."
7. Define: "maximum breathing capacity."

8. What is the normal vital capacity?
9. What is the normal maximum breathing capacity?
10. What does a reduction of vital capacity below 2.5 liters indicate?
11. If the maximum breathing capacity is above 100 liters, what does it indicate?
12. If the maximum breathing capacity is below 50 liters, what does it indicate?
13. In evaluating cardiac function, what tests and physical signs are used?
14. Can surgery be done in the presence of myocardial infarction? Explain.
15. What are the cardiac contraindications to pulmonary surgery?
16. Discuss the evaluation of the following: right ventricular hypertrophy, right heart strain, right bundle block.
17. Discuss the importance of kidney function in thoracic surgery.
18. When is surgery contraindicated in the presence of abnormal kidney function? What tests would you use?
19. Discuss the importance of liver function in thoracic surgery.
20. Discuss the role of the anesthetist in thoracic surgery.
21. Give the treatment of cardiac arrest.
22. Give the treatment for ventricular fibrillation.
23. Describe the essential care before the patient is moved out of the operating room.

CHAPTER 36

SURGERY OF THE CHEST—THORACIC INCISIONS

By George W. Holmes

The type and choice of thoracotomy incision will depend upon the following factors: the location of the lesion in reference to the thoracic cage, the pathological nature of the lesion, *i.e.*, whether benign or malignant and the extent and scope of the contemplated surgical procedure.

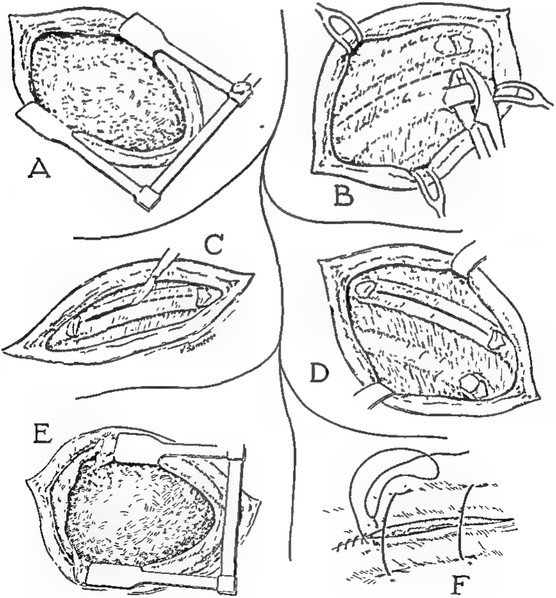

FIG. 220.—Diagrammatic il[illegible]

[illegible] approximating

Unlike an abdominal incision, downward and upward retraction of a thoracotomy incision is not possible. For this reason the thoracic incision must be planned and made so that the widest exposure is obtained to the underlying structure to be surgically dealt with. Obviously, this requires preoperative localization of the lesion, its nature, and extent of the contemplated surgical procedure.

POSTERIOR INCISION

The patient is in a prone position on a special operating table attachment which suspends the head, upper extremities and chest from the rest of the body.

The shoulder is manipulated to rotate the scapula outward as much as possible. A parascapular incision is started at the level of the 2nd rib midway between *the vertebral border of the scapula and the dorsal spine. The incision curves down* and around the inferior angle of the scapula and then along the course of the rib to be resected. The finger of the left hand can be slipped through the triangle of

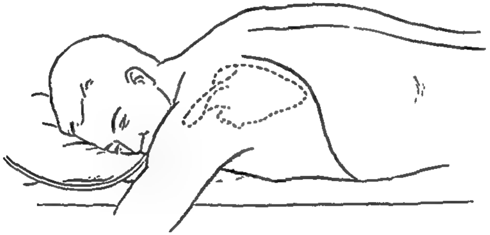

FIG. 221.—Position of the patient for the posterior incision.

auscultation with minimal blunt dissection and in this way the trapezius and rhomboid muscles can be lifted up with the left fingers and the muscle fibers incised with the scalpel. As the scapula is elevated and rotated laterally, the loose areolar tissue attaching the scapula to the chest wall can be separated bluntly. Further mobility of the scapula is accomplished by elevating the latissimus dorsi and inferior angle of the scapula attached to the scapula. Cutting muscle fibers of the levator scapula permits further lateral retraction exposing the posterior half of the upper six ribs. The latissimus dorsi and serratus anterior muscles are retracted laterally with the scapula to expose anterior segments of the upper six ribs.

After mobilization of the scapula, the next step is the resection of the rib; for pneumonectomy the entire 4th rib is usually resected and for a lower lobectomy the entire 6th rib. An incision is now made over the longitudinal midportion of the designated rib and the periosteum peeled upward and downward. The rib is removed by subperiosteal resection using a periosteal elevator. Subperiosteal resection of the rib is associated with less bleeding than extra-periosteal resection of the rib. Using bone cutting forceps the rib is cut off at the lateral process of the spine and anteriorly at the anterior axillary line. After subperiosteal resection of the rib, the anterior and posterior intercostal arteries are ligated. In order to obtain

greater exposure, a segment of rib above and below are excised in a similar manner. The subperiosteal bed of the rib resected is incised in the midline and the pleural cavity is opened.

Care must be used in opening the parietal pleura to make certain that it is free and not obliterated. An obliterated pleural cavity requires time and careful dissection of the adhesions before the ribs can be spread by a mechanical rib spreader. It is important to incise the intercostal space or the divided subperiosteal bed both anteriorly and posteriorly for some distance before using the mechanical rib spreader in order to prevent rib fractures.

A free pleural cavity makes the intrathoracic surgical procedure much easier. The obliterative pleural cavity requires time and considerable blood loss before the lung is mobilized. Extreme apical and extreme diaphragmatic adhesions are usually the most difficult to dissect. The right upper mediastinal space is the most dangerous during the dissection because of injury to the thin innominate vein, superior vena cava or azygos vein.

Anesthesia is relatively easy in this position except that the anesthesiologist must sit on a low stool and anchor the endotracheal tube securely so that it does not slip out of the air passages. The surgeon now has splendid exposure to the posterior mediastinum and to the main bronchus and its branches. In those cases where there is pulmonary bleeding or suppuration, the surgeon is in a position to clamp the main bronchus or the lobar bronchus and allow the anesthesiologist to aspirate the air passages until clear. The exposure of the anterior mediastinum is poor. It is difficult to retract the lung over to expose the anterior mediastinum for dissection of the hilar vessels. If the main bronchus or the lobar bronchus is divided the artery and vein can be dissected out.

Closure of the incision, after insertion of water-seal catheters, requires approximation of the ribs while the transgressed subperiosteal bed is closed with a continuous or interrupted suture. Catgut or wire sutures around the adjacent ribs helps to maintain rib approximation. All chest wall muscle layers are approximated with continuous or interrupted sutures. With good rib approximation and good wound healing, the postoperative disability is moderate and chest wall pain may persist for some time. Physiotherapy and early motion of the arm on the operated side is important.

LATERAL INCISION

With the patient in the lateral position and the operative side of the chest leaning slightly forward, a parascapular incision is made similar to that described for in the posterior incision. The patient's position is maintained by anchoring the patient's pelvis to the table with wide adhesive tape and sand bags. As contrasted to the posterior incision, the lateral incision can be extended to the anterior axillary line.

The more anterior the rib resection is carried, the greater the thoracic exposure after the rib spreader has been used to its fullest capacity. This incision affords the best exposure and is more universally used. All parts of the mediastinum are visible and the deflated lung can be manipulated into any position during the hilar dissection. Bleeding from pleural adhesions, mediastinal adhesions, or hilar dissections is more easily controlled through this exposure. The anesthesiologist must assist the patient's respiration by manual manipulation or mechanical means because expansion of the contralateral chest is limited in this position. Also, the

"shifting" mediastinum must be maintained during respiratory exchange and aspiration of the air passages. The chance of hypoxia, anoxia, or hypercarbia is much greater than in the anterior or posterior incisions, but anesthesiologists are becoming more skilled in recognizing and correcting such conditions.

After the thoracic surgery has been completed, the closure of the chest wall is carried out in the same manner as described for closure of the posterior incision. Probably the main advantage of the subperiosteal resection of a rib instead of the intercostal incision is less bleeding and allows a more firm and secure suture closure so that the heavy catgut or wire sutures around the adjoining ribs can be eliminated. If the intercostal or subperiosteal incision is not securely closed, a gradual separation of the ribs takes place with resulting postoperative pain.

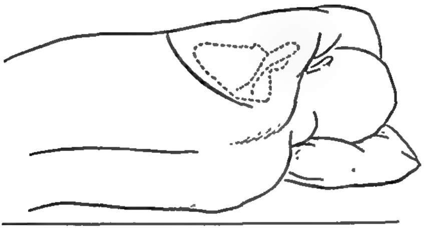

FIG. 222.—Position of patient for lateral incision.

ANTERIOR INCISION

With the patient in the supine position a small sand bag is placed under the shoulder and hip of the side to be operated. The arm of the same side is abducted on an arm board or suspended from a stand in a semi-flexed position. The arm should not be abducted too far in order to prevent a "stretch" palsy of the brachial plexus.

The incision is made from the midline of the sternum to the mid-axillary line in the anterior 4th interspace. After hemostasis of the skin and subcutaneous tissues, the lateral border of the pectoralis major is exposed and by blunt dissection it is freed so that the left index finger can be passed under the muscle. The muscle fibers are then cut along the 4th interspace to the body of the sternum. The costochondral junctions of the ribs are exposed and a small segment of the 4th and 5th rib cartilage is resected, sub-perichondrally just medial to the costochondral junction. The 4th interspace is then opened; care being used until the presence of a free pleural cavity or obliterative pleural cavity is determined. The interspace incision is carried forward and then up and down through the subchondral bed of the resected cartilage (4th and 5th). The incision should not be carried anterior beyond this or the internal mammary vessels may be injured. Usually the anterior intercostal arteries from the internal mammary artery are severed and must be ligated after the rib spreader is inserted. The incision is opened laterally by further incision of the 4th interspace and division of the pectoralis minor and other axillary areolar tissue.

This incision is simple and preferred by the anesthesiologist because of ease of maintaining anesthesia and for aspirating the tracheobronchial tree. The surgeon has splendid access to the anterior mediastinum and the anterior portion of the pulmonary hilum, but exposure of the posterior mediastinum is poor and the incisional opening is not adequate to retract the lung over anteriorly to examine the posterior mediastinum. This exposure is ideal for dissection of the intra-hilar structures.

Closure of the incision, after placing of water-seal catheters, requires heavy catgut or wire sutures to approximate the ribs to their normal position and to approximate the costochondral junctions. The intercostal muscles, pectoralis major and minor are closed in layers with interrupted or continuous sutures. Subcutaneous tissue and skin edges are closed with interrupted sutures. It is to be noted

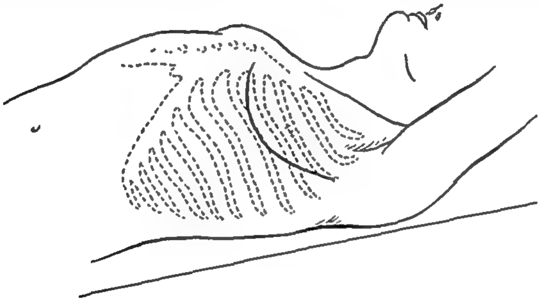

FIG. 223.—Position of patient for anterior incision.

that the muscle layer closure covering the intercostal chest incision is thin compared to other incisions into the thorax and must be approximated securely and evenly for good wound healing. With firm rib approximation and good wound healing, the postoperative disability of this incision is minimal.

THORACOABDOMINAL INCISION

An anterior thoracic incision made in the 5th or 6th interspace may be carried downward dividing the costal arch and diaphragm, and then combined with an upper paramedian abdominal incision. This allows exposure to the lower chest and upper abdomen. The chest is closed in the usual way with emphasis on secure approximation of the chondral arch. The diaphragmatic and abdominal incisions are closed in the usual manner.

The lateral incision with resection of the 6th or 7th rib may be carried anterior and the costal arch and diaphragm divided and the incision connected with an upper paramedian incision. Again, this allows exposure of the entire chest and upper abdomen. The chest is closed in the usual manner and the incision in the

anterior diaphragm is closed with interrupted silk sutures. The abdominal incision closure is done in the usual manner.

QUESTIONNAIRE

1. What factors influence the type and choice of thoracotomy incision?
2. In what respects does a thoracotomy incision differ from an abdominal incision?
3. Describe the technique for a posterior incision.
5. Describe the technique for a lateral incision.
6. Describe the technique for an anterior incision.
7. Describe the technique for a thoracoabdominal incision.

CHAPTER 37

SURGERY OF THE CHEST—OPERATIVE PROCEDURES

By George W. Holmes

PNEUMONECTOMY

Complete resection of the lung or pneumonectomy is done only for specific indications. Obviously, the contralateral lung must be near normal to carry an adequate respiratory function after completion of the surgery. Likewise, the contralateral lung must be maintained in the "position of rest" during and after surgery. Indications for pneumonectomy are: acute multiple lacerations of the lung due to shrapnel wounds as occur during wartime, primary neoplasm of the lung limited to the lung and draining hilar lymph nodes; partial to complete bronchial occlusion due to disease with chronic infection in the lung parenchyma distal to the bronchial obstruction; and completely destroyed lung parenchyma due to disease with or without bronchial stenosis. There are occasional other indications but these will vary with the individual case and the experience of the surgeon.

The contraindications of pneumonectomy are: disease or respiratory insufficiency in the contralateral lung; chronic renal or cardiac disease which would be aggravated and become irreversible by the surgery; neoplastic lung disease which has extended beyond the draining hilar lymph nodes into the abdominal cavity or into the supraclavicular fossa; neoplastic disease which has invaded the phrenic nerve, recurrent, laryngeal nerve or mediastinal vessels.

Technique: Left Pneumonectomy-anterior Approach.—A left pneumonectomy can be accomplished through any one of the thoracotomy incisions described. However, anterior approach is preferred by the writer because it causes the least disturbance to the cardio-pulmonary functions during anesthesia.

After the anterior incision has been made on the left side of the chest, and the pleural cavity opened, any adhesions between the lung and chest wall are cut near the visceral pleura. This allows the adhesion to contract to the parietal pleura and if some bleeding continues from the ends of the adhesions they can be grasped with a hemostat and ligated. The inferior pulmonary ligament is freed from the diaphragm to the inferior pulmonary vein by blunt dissection with the left index finger. With the lung fully mobilized, the extent of the pathology is determined in relation to the hilar vessels and the bronchus. The surgeon must determine if the vessels and bronchus can be adequately mobilized, securely ligated, divided and adequate bronchus is available for a good closure. The mediastinal pleura is opened by blunt dissection over the hilum and just posterior to the phrenic nerve. The mediastinal pleura is opened to the inferior side of the aorta and downward to the inferior pulmonary vein. A one-centimeter segment of dental roll held by an 8-inch hemostat is used as a dissecting-sponge instrument. With the dissecting-sponge, the superior pulmonary vein and pulmonary artery are stripped of small lymph nodes, fatty tissue and folds of pleura. The superior and inferior edges of

the superior pulmonary vein are freed and when 75 per cent of the circumference of the vein is exposed, a Rumel dissecting hemostat is passed under it and emerges on the opposite side. A 2-0 silk suture (18 to 30 inches long) is placed in the slightly open jaws of the Rumel clamp and pulled under the vein for ligation. This ligature is placed as near the mediastinal pleura as possible and tied.

The superior pulmonary vein is then stripped laterally until it bifurcates into the segmental veins. Each segmental vein is identified and ligated with 2-0 silk suture with the aid of the Rumel dissecting hemostat. After all segmental veins are

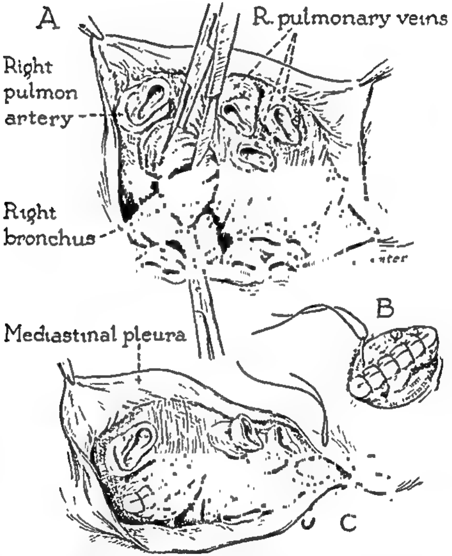

FIG. 221.—Steps of pneumonectomy: (*A*) ligation of pulmonary artery and vein and division of main bronchus; (*B*) closure of bronchus; (*C*) covering of hilum structures with mediastinal pleura.

tied, there is approximately 1½ cm. of vein between the proximal and the distal ligatures. The vein is divided over an instrument to protect the underlying artery. The proximal end of the vein is picked up with a hemostat and ligated with a transfixion suture. As the proximal stump of the doubly ligated superior pulmonary vein retracts into the mediastinum, the inferior pulmonary vein is seen just below.

To expose the inferior pulmonary vein, it becomes necessary to cover the pericardium with a lap pad and retract the pericardium and heart medially with a retractor. Great care must be used not to obstruct the left ventricular output or

irritate the myocardium with the retractor. Any cardiac irregularity indicates that the cardiac retraction is too forceful. With the heart retracted medially, a better view of the inferior pulmonary vein is obtained. The vein now is stripped clean with the dissecting sponge until 75 per cent of the circumference of the vein is visible. The Rumel clamp is passed around the vein which is then ligated as proximally as possible. The segmental veins are dissected and ligated individually. After an adequate stump is obtained on the proximal side, the vein is cut and the proximal stump is transfixed and ligated.

Immediate ligation and division of the veins prevents embolization of the tumor or infection into the left auricle. Contrary to some reports, immediate division of the pulmonary veins does not cause arterial engorgement of the lung. With the lung retracted laterally the pulmonary artery is stripped clean on its anterior, superior, and posterior surfaces as far as possible. The vagus nerve is seen posterior to the hilum and is dissected carefully from the posterior surface of the pulmonary artery. Dissection of the pulmonary artery is carried out until 75 per cent of its circumference is free and then a Rumel curved hemostat is passed the rest of the way around the artery. Again a 2-0 single or double silk suture is passed around the artery and securely ligated. The artery is stripped distally until the segmental arteries to the left upper lobe are identified. These are ligated at the point of origin from the main stem artery and divided distally. As the segmental lobar arteries are divided, the entire main stem artery comes into view and is further dissected out so that it can be transfixed and ligated at least 1 centimeter distal to the proximal ligature. The artery is then divided leaving a generous cuff on the proximal side. The distal stumps of all vessels are ligated *in situ* before division to prevent the troublesome bleeding from the distal vessel openings. All vessels are doubly ligated on the proximal side with transfixation of the more lateral ligature. At least one centimeter of vessel cuff remains between the ligatures to prevent slipping during increased mediastinal shifting.

The lung is now retracted further laterally and all the bronchial glands from the carina outward are stripped laterally until 2 to 4 centimeters of the main bronchus is exposed from the carina. The bronchus is divided and the lung and hilar glands removed as one specimen.

The bronchial stump may be closed by the open or closed method. In the open method, the bronchus is divided so that the membranous portion is longer than the cartilage rings. The open lumen of the bronchus is suctioned out and the membranous portion is sutured to the cartilage rings with interrupted 3-0 silk sutures. The sutures are placed along and behind the cartilaginous bronchial ring and grasping a good bite of the membranous portion until the bronchial closure is complete. In the closed method, the bronchus is grasped with a strong bronchial clamp and divided sufficiently distal to the clamp so that a cuff of the bronchus can be sutured with a series of interrupted sutures. The bronchial clamp is then removed and a second row of mattress sutures is inserted for re-enforcement.

Wherever possible the bronchial stump is covered with a flap of parietal or mediastinal pleura or a flap of mediastinal pleura containing pericardial fat. This aids the healing of the bronchial stump but is not an absolute guarantee that the bronchus will heal without fistula formation. Healing is more closely related to the patient's general cellular nutrition and nitrogen balance and adequate antibiotic therapy.

After bronchial closure, the thoracic cavity is filled with normal saline and the

anesthesiologist exerts endotracheal pressure up to 20 cm. of water by compressing the rubber bag in the closed anesthesia system. Any air leak from the bronchial stump indicates incomplete closure and thus requires more sutures. This is repeated until the bronchial closure is tight and no air leaks are seen. The chest cavity is washed out with normal saline until the aspirated saline is relatively clear or only slightly sanguineous. Usually 10,000,000 units of penicillin and 3 gms. of streptomycin are placed in the chest cavity before a tight closure of the thoracic cage is carried out. Air is aspirated from the operated chest with a large syringe (50 ml.) and aspirating needle (# 15) until a negative pressure is observed by suction of the syringe barrel. A small dressing is applied so that respiration of the contralateral chest is not handicapped.

LOBECTOMY

The indications for lobectomy are too numerous to outline. A chronic inflammation or tumor, localized to one lobe of the lung may be resected by lobectomy. If frozen section of the lesion reveals a tumor, then the remaining portion of the lung can be resected with the hilar lymph nodes.

Technique: Right Upper Lobe Lobectomy, Anterior Approach.—*After* the right anterior thoracotomy incision has been made, the pleural cavity is examined for adhesions. In the case of inflammatory disease, adhesions between the diseased lobe and parietal pleural usually are present. If the lung is closely adherent to the chest wall, it may be advisable to do an extrapleural stripping of the lung and adherent parietal pleura and then enter the pleural cavity as soon as a free space is encountered. Lysis of adhesions existing between the right upper lobe and the mediastinum must be done with care in order to avoid injury to the right innominate vein, azygos vein, superior vena cava, the phrenic nerve and the vagus nerve. The inferior pulmonary ligament is divided under direct vision up to the inferior pulmonary vein. During division of this ligament any aberrant artery is doubly clamped, divided and both ends ligated. Hilar exposure is obtained by opening the mediastinal pleura posterior to the phrenic nerve and over the surface of the superior pulmonary vein. The anterior surface of the vein is stripped to the pericardium and laterally to the segmental divisions. The vein draining the right upper lobe is identified from the vein draining the right middle lobe. Superior and inferior edges of the vein draining the right upper lobe are exposed, and after 75 per cent of the vein circumference is exposed, a curved forceps is passed by blunt dissection under its posterior surface. The vein is found closely adherent to the underlying artery to the right middle lobe and right lower lobe.

After completing the dissection, the vein is ligated with a silk suture (2-0). The segmental veins draining the apical and anterior pulmonary segments are dissected free, ligated and divided. After these segmental veins are divided the posterior segmental vein to the right upper lobe is seen and can be easily dissected free and ligated and divided The medial stump of the superior pulmonary vein to the right upper lobe is transfixed and ligated. C- upward, the superior pulmonary artery to the right upper lobe is now is stripped clean until 75 per cent of the artery circumference is expo hemostat is passed under the artery by blunt n The artery to the underlying right upper lobe b he artery is li sutures (2-0).

lobe; it passes posterior and toward the segmental branches of the right upper lobe bronchus. It is carefully dissected and a forceps is passed under it. It is ligated and divided at its origin from the main stem inferior pulmonary artery.

The pulmonary attachment between the apex of the right lower lobe and right upper lobe is divided between clamps and the lung tissues under both clamps are transfixed and ligated. The right upper lobe bronchus is exposed with the lobe retracted laterally. The segmental bronchi are identified and divided between clamps. After the lobe is removed, the segmental bronchi are opened and the spurs between are resected. The bronchial lumen is converted into one large opening and closed with interrupted silk sutures (3-0) obtaining a good "bite" of the anterior and posterior bronchial walls. Three mattress sutures are placed between the distal row of sutures and the main bronchus to further secure closure of the right upper lobe bronchial stump. A flap of mediastinal or parietal pleura is reflected and sutured over the bronchial stump with several interrupted sutures.

The chest cavity is cleansed with sterile water or saline and the remaining lung is inflated up to 15 cm. of water pressure on the closed anesthesia system. Minor air leaks causing bubbles may be seen coming from the sutured interlobar fissure area sometimes requiring further sutures. Two water-seal mushroom catheters are inserted; one in the anterior first mid-clavicular space and the other in the axillary line, 5th or 6th interspace. The anterior thoracotomy incision is closed in the usual way.

SEGMENTAL LOBECTOMY

Segmental lobectomy is reserved for the more localized chronic infections or tumors, when a resection such as lobectomy is unnecessary.

Technique: Basilary Segmental Resection, Lower Lobe, Anterior Approach.—The usual anterior thoracotomy incision is made. The lower lobe is freed by cutting of all peripheral adhesions. The inferior pulmonary ligament is divided up to the inferior pulmonary vein. The entire vein is dissected free so that a curved hemostat can be passed around it easily. The vein is then dissected laterally until it bifurcates into three or four segmental branches. The branch to the apex of the lower lobe and passing posterior to the lower lobe bronchus is preserved. The other segmental veins are ligated at the bifurcation of the inferior pulmonary vein. The ligated segmental veins are dissected laterally and divided. The major fissure is dissected back to the hilum and by retracting the upper lobe into the upper chest, the pulmonary artery to the lower lobe is seen adherent and lateral to the bronchus.

The lower lobe pulmonary artery is carefully dissected free from the bronchus and ligated just before it bifurcates into the anterior and posterior basilar branches. The segmental artery branches are dissected laterally, ligated and divided. The basilar segmental bronchus to the lower lobe is dissected on all sides and distally to the bifurcation of the anterior, lateral and posterior branches. Each are doubly clamped with hemostats and divided. With clamps on the distal ends of the basilar segmental bronchi, arteries, and veins, the surgeon is ready to make an artificial fissure and separate the basilar segment of the lung from the apical segment. The distal ends of the arteries and veins are dissected laterally as well as the segmental bronchi until the visceral pleura is approached from the inside of the lung tissue. The visceral pleura is then incised parallel to the base of the lower lobe and usually 1 to 2 cm. above the edge. This procedure is best done with the lung partially inflated as the apical segment is distended and the basilar segment is

atelectatic, thereby making the line of artificial fissure somewhat easier to recognize. After removal of the basilar segment, the underside of the apical segment appears raw and ragged. There will be bleeding from small interlobular veins which must be meticulously ligated, as well as any bronchopleural fistulæ that are recognized. The basilar segmental bronchus is opened and converted into one bronchus by resecting out the segmental branch spurs. Retrograde aspiration of the open bronchus is done and the bronchial stump closed with interrupted silk sutures (3-0). Proximal mattress sutures may be placed if they do not encroach on the apical segmental bronchus. The stump is then covered by a reflected flap of mediastinal or parietal pleura.

The chest cavity is cleansed with sterile water or saline and the lung inflated. The bronchial stump is held under water and examined for bubbles while the lung is inflated with a 15 cm. water pressure. Any bubbles coming from the raw surface of the apical segment must be stopped by ligatures. After all bleeding has been controlled de Pezzer catheters (# 32) are inserted in the anterior first interspace and 5th or 6th interspace axillary line and attached to the water-seal bottles. Closure of the incision is done in the usual fashion.

PULMONARY WEDGE RESECTION

A wedge resection of the lung is done when a small biopsy of the lung is desired or when a small nodule is located peripherally and does not require a segmental lobectomy or lobectomy for its removal. If the frozen biopsy of the tissue proves to be malignant, lobectomy or pneumonectomy can be carried out.

Technique.—An anterior incision is made in the usual way. After the pleural cavity is opened, peripheral adhesions between the lung and chest are divided and the lung is mobilized in the area of pulmonary disease or nodule which is to be resected. Carmel clamps are placed so that a triangular-shaped segment of the lung containing the pathology is resected between the clamps. The lung tissue is sutured with a running catgut suture under each clamp and pulled as the clamp is removed. The cut edge of the lung is folded up accordion style and the catgut tied. If hemostasis is not complete a few interrupted sutures may have to be placed to stop the bleeding. The chest cavity is cleansed with sterile water or saline and the lung inflated to test the rows of catgut suture for any air leaks. If none, the water-seal drainage tubes are placed as for lobectomy or segmental lobectomy and the chest closed in the usual way.

DECORTICATION OF THE LUNG

Decortication of the lung is indicated whenever hemothorax persists after multiple chest aspirations. The ideal time for this procedure is between the fourth and sixth week following the causative injury and hemothorax. Decortication can also be done in cases of chronic pleural effusion and empyema. The object of the operation is to free the captive lung and improve its respiratory function.

Technique.—Incision of the chest is made through the lateral or anterior approach. The former approach gives the largest exposure to the chest cavity. Rarely is a free pleural cavity encountered. However, in the apical area the pleural thickening is not as marked as in the lower chest. The parietal pleura is incised care-

fully until the pigmented visceral pleura is seen. The visceral pleura and atelectatic lung are dissected from the thickened parietal pleura and if the dissection takes place in the right plane, the procedure is like peeling an orange. Care must be used in following the cleavage plane as it is very easy to lacerate the visceral pleura resulting in multiple bronchopleural fistulæ.

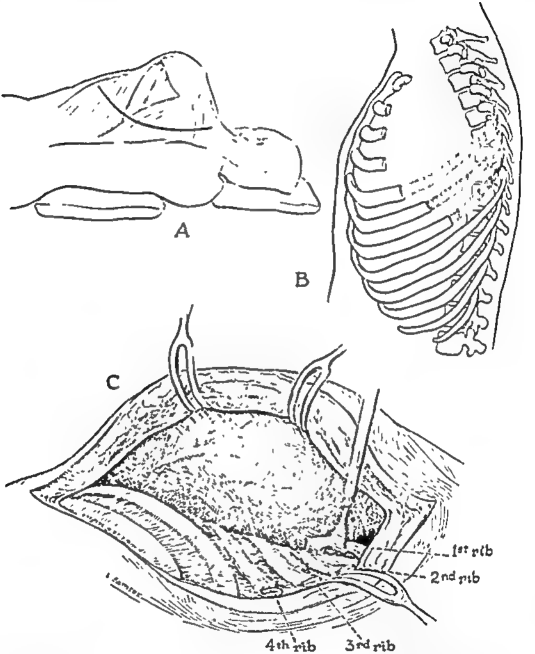

FIG. 226.—Steps of thoracoplasty operation; (*A*) incision; (*B*) segments removed at first stage. Shaded segments to be removed at second stage; (*C*) exposure with elevation of scapula.

The "peel" must be dissected away from the atelectatic lung and diaphragm until upon inflation of the lungs, all lobes are fully expanded and the diaphragm moves freely. If a portion of the lung is not adherent to the parietal pleura, the procedure is easier as the surgeon can procede downward from the free pleural cavity and find the cleavage plane of dissection. The chest cavity is cleansed with sterile water or saline while the lung is inflated. Large bronchopleural fistulæ are closed with interrupted purse-string sutures on a small needle. The fistulæ may be

clamped with a small curved hemostat and ligated. Two de Pezzer catheters are placed before closure of the chest and attached to the water seal bottles. The chest closure is done in the usual manner and the lung is kept inflated during the closure.

Following surgery one water-seal catheter may be attached to constant suction. Every effort is made to keep the lung inflated and adherent to the chest wall so as to prevent a postoperative hemothorax from occurring.

THORACOPLASTY

The thoracoplasty operation was once the most common thoracic surgical procedure. It is still employed for the collapse of upper lobes containing open tuberculosis cavities that have failed to respond to other forms of medical therapy. It is a procedure used to obliterate a chest cavity following pneumonectomy or to obliterate a chronic empyema space. Because of the postoperative deformity of the chest, artificial prosthesis such as paraffin wax, Lucite balls, and Ivalon sponge have been placed over the periosteal rib beds to minimize the deformity due to the sagging of the scapula.

Technique.—A lateral thoractomy approach is made under endotracheal or local anesthesia. Subperiosteal resection of the fourth, third, second, and first ribs are carried out from the transverse process of the vertebræ to the costo-chondral junction or mid-clavicular line. Further segments of the fifth, sixth and seventh ribs are resected if necessary. It is seen that the lung under the subperiosteally resected ribs has collapsed and paradoxical motion is present during respiration. At this time, sterile paraffin wax, sterile Lucite balls, or sterile Ivalon sponge is placed over the area of resected ribs. The scapula is pulled back into position and the wound is flooded with an antibiotic solution. The muscles and skin edges are closed with interrupted silk sutures. Unless the paraffin wax, Lucite balls or Ivalon sponge was inserted under the scapula before the wound is closed there would be a marked deformity with the scapula sagging into the area of the resected ribs. This deformity was a serious objection to the thoracoplasty operation before the modification of using a prosthesis under the scapula (*see* Fig. 226).

CHEST WALL RESECTION

Any chest wall tumor or chronic osteomyelitis can be resected *in toto*. It is often necessary to resect the rib above and below in order to get the lesion out *en bloc*. This leaves a large sucking wound of the chest wall but it can be closed by closing the defect in the chest wall with sterile stainless steel mesh or tantalum wire mesh sutured to the edges of cut intercostal tissue and ribs. This gives the chest wall rigidity, and the chest wall muscles and skin cover the wire. One or two water seal drainage bottles are necessary for the re-expansion of the lung.

QUESTIONNAIRE

1. What [illegible]
2. [illegible]
3 [illegible]
4. [illegible] of left pneumonectomy.

5. What are the indications for lobectomy?
6. Describe the technique for right upper lobe lobectomy.
7. What are the indications for segmental lobectomy?
8. Describe technique of basilary segmental resection.
9. When is a pulmonary wedge resection indicated?
10. Describe the technique for a pulmonary wedge resection.
11. When is decortication of the lung indicated?
12. Describe the technique for decortication of the lung.
13. Discuss the role of thoracoplasty in present day thoracic surgery.
14. What is its purpose?
15. Describe the technique.

CHAPTER 38

SURGERY OF THE CHEST—SURGERY OF THE DIAPHRAGM

By George W. Holmes

Since rhythmical contraction of the diaphragm is such an intricate part of each respiratory cycle, surgery on the diaphragm is not contemplated unless the diaphragmatic efficiency can be improved. If the diaphragm must be transected for some thoraco-abdominal surgical procedure, the incision is limited to the fibrous central tendon. Every effort is made to preserve the muscular portion or as much of it as possible. Also, the diaphragm serves as the main partition between the thoracic and the abdominal cavity; consequently it is subject to many pressures from the thoracic and abdominal cavities. Surgery of the diaphragm is indicated for herniations, eventration, injury, or tumors, of the diaphragm.

SLIDING HIATAL HERNIA

This is the most common diaphragmatic hernia and is reported often by the radiologist during upper gastrointestinal tract study with barium contrast media and often by the surgeon during exploration of the abdomen. Anatomically, there is a peritoneal lined sac which extends into the mediastinum posterior and laterally to the left side of the esophagus and displaces the left crura laterally. The cardia of the stomach slides into the sac on increased intra-abdominal pressure. In time and with further increased intra-abdominal pressure, the esophago-gastric junction and the posterior esophageal ligaments slide into the sac. Radiographically, the esophagus appears shortened and gives the impression that a short esophagus is responsible for pulling the stomach up into the mediastinum. Actually during surgery the esophagus is redundant on itself and not shortened unless by previously acquired esophagitis with fibrous shortening of the esophagus.

PARA-ESOPHAGEAL HERNIA

The para-esophageal hernia must be differentiated from a sliding hiatal hernia because the peritoneal sac enters the mediastinum lateral to the crura. The crura is intact and the esophageal hiatus is not enlarged. Radiographically, the esophagus is of normal length and the esophago-gastric junction is below the diaphragm but the barium goes down into the stomach and then up into the hernial sac containing fundus of the stomach above the diaphragm and adjacent to the esophagus. The barium contrast column is referred to as a hook or J-formation on radiograms.

Technique.—A left anterior thoractomy incision is made in the sixth or seventh interspace depending on the A-P diameter of the lower chest. The ribs are divided at the chondral arch, but the arch is maintained intact. The sixth or seventh intercostal space is opened and the rib spreader inserted. A good view of the diaphragm is obtained; the central tendon is incised and the index and middle fingers are

passed under the diaphragm into the sac; with the fingers in the sac the position of the left crura is determined. The surgeon can now differentiate the para-esophageal from the hiatal hernia and determine its exact size and the relation of the sac to the esophagus. The central tendon is opened radially so that the surgeon can get his entire hand under the diaphragm, reduce the hernia, and hold the stomach down. In the case of the para-esophageal hernia, the peritoneal sac above the diaphragm is resected and the opening in the diaphragm adjacent to the left crura is closed with interrupted silk sutures.

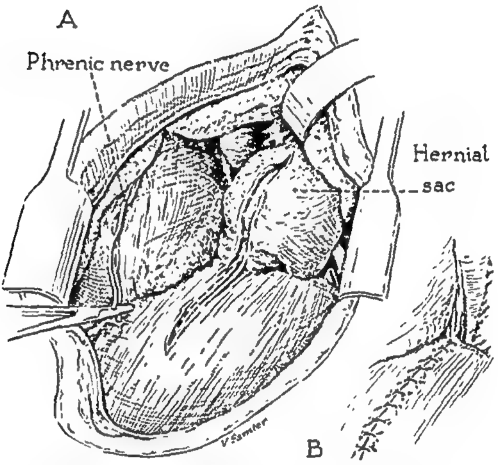

FIG. 227.—Transthoracic repair of diaphragmatic hiatal hernia: (A) opening of diaphragm and hernial sac, (B) closure of defect after reduction and resection of sac.

In the case of a hiatal hernia, the radial incision of the central tendon is continued through the muscle of the left crura. The lower end of the esophagus and the esophago-gastric junction are mobilized. The peritoneal sac is resected peripherally as well as the esophageal ligaments so that there is a collar of peritoneum around the mobilized esophago-gastric junction. The posterior muscle of the left crura is sutured behind the esophagus to the right crura with several interrupted silk sutures, thereby closing the original hiatal opening.

The esophagus is then transplanted slightly anterior to the normal position. The central tendon is closed with interrupted silk sutures, and the new esophageal hiatal opening is made so that there is enough room to insert the little finger through the diaphragmatic opening alongside the esophagus. In both the para-esophageal and hiatal diaphragmatic hernia repair, the peritoneal sac and ligaments attached to the esophago-gastric junction are deliberately included in the sutures closing

the central tendon. By this maneuver the esophago-gastric junction is well anchored by sutures under the diaphragm. After closure of the central tendon, the diaphragm is seen to be functioning normally. One water-seal catheter is inserted in the lower axillary line, and the thoracotomy incision closed in the usual manner.

CONGENITAL HERNIA

Hernias through the foramen of Bochdalek or Morgagni or through a congenital defect of any portion of the diaphragm are usually seen in infants and children. Repair may require closure of the defect with fascia lata, tantalum mesh, or transplanted chest wall muscle. The thoracic surgical incision will depend on the location of the hernia in the chest cavity.

ACQUIRED HERNIA OF FORAMEN OF MORGAGNI

The anterior diaphragmatic hernia through the foramen of Morgagni is the rarest acquired diaphragmatic hernia in the adult. It is usually asymptomatic and must be differentiated from a pericardial cyst or dermoid cyst. It is usually discovered in routine chest radiograms. If the exact diagnosis can be established the repair of the hernia can be done without thoracotomy.

Technique.—An upper mid-line abdominal incision is made and the properitoneal fat exposed. This fat is seen to extend upward under the posterior wall of the rectus abdominis muscle and pushes the anterior portion of the muscular diaphragm laterally and occupies the anterior mediastinum. A sac of peritoneum may also be pulled up by the properitoneal fat into the anterior mediastinum with loops of small and large bowel in the peritoneal sac. After reducing the hernia, the excessive properitoneal fat can be amputated and the peritoneal sac inverted and closed. The muscular portion of the anterior diaphragm is pulled toward the midline and sutured to the posterior sheath of the rectus abdominis muscle. The surgeon must be careful not to tear or perforate the mediastinal pleura. The midline incision is closed with interrupted sutures.

TRAUMATIC DIAPHRAGMATIC HERNIA

These hernias are seen more often today due to frequent and severe automobile accidents. The tear in the diaphragm can involve the muscular portion or both the muscular and central portions. Much of the abdominal viscera can slide through into the thoracic cavity causing compressive atelectasis of the lung. The abdominal viscera becomes adherent to the lung, chest cavity and mediastinal pleura and can become incarcerated or obstructed. Early operative reduction of the hernia with diaphragmatic repair is indicated before troublesome adhesions or pleural effusion complicate the surgery.

Technique.—The anterior or lateral thoracotomy incision can be used. Good exposure is essential for the meticulous dissection of the viscera adherent to the lungs, mediastinal pleura and exposed surfaces of the lacerated diaphragm. After reduction of the hernia, the tear in the diaphragm is closed with interrupted sutures. Decortication of the lung may be necessary to obtain adequate expansion. Following repair of the diaphragmatic hernia abdominal bowel obstruction must be considered and may require surgical correction.

TUMORS AND CYSTS OF THE DIAPHRAGM

Isolated benign tumors or cysts of the diaphragm should be resected and the defect in the diaphragm repaired with tantalum mesh or fascia lata. The lateral thoracotomy incision is best for this type of surgery.

EVENTRATION OF THE DIAPHRAGM

If the diaphragm becomes stretched and thinned out its effective motion for respiration becomes inadequate. The diaphragm becomes elevated and permits the abdominal viscera to move up into the thoracic cavity. This may lead to bronchial kinking and pulmonary atelectasis. The ineffectual diaphragmatic motion leads to a weak cough and permits secretions to collect in the dependent bronchus and results in a chronic pneumonitis. Abnormal vago-vagal reflexes have been described when the viscera slides up into the thoracic cavity. In such cases, surgery for the eventration must be done.

Technique.—Through a lower lateral thoracic incision, the entire diaphragm is exposed. It is seen to be thin and practically motionless. The peripheral musculature shows weak rhythmical contractions. By pushing down on the diaphragm the abdominal viscera is also pushed down into the abdomen. The redundant thin diaphragm can be grasped like the skin of a dog's back and folded over on itself and securely imbricated and sutured so that it can withstand the increased intra-abdominal pressures. It will be seen that the diaphragmatic motion is somewhat increased and assumes a normal position in the thoracic cavity. Closure of the chest is carried out in the usual manner.

QUESTIONNAIRE

1. What are the general indications for surgery of the diaphragm?
2. What is the most common hernia of the diaphragm?
3. Give the surgical anatomy of a hiatal hernia.
4. What is the difference between a hiatal and a para-esophageal hernia?
5. What is the roentgenographic appearance of a para-esophageal hernia?
6. Describe the technique for the repair of these herniæ.
7. What is a congenital hernia of Morgagni?
8. How is the repair of such hernia accomplished?
9. Discuss traumatic diaphragmatic herniæ.
10. Discuss eventration of the diaphragm.

CHAPTER 39

SURGERY OF THE CHEST—SURGERY OF THE ESOPHAGUS

By George W. Holmes

Absence of a serosa on the esophagus excludes surgery of the esophagus as part of gastro-intestinal surgery. The esophagus can be studied completely by contrast barium study and esophagoscopy; only lesions at the esophago-gastric junction must be differentiated by surgical exploration. The esophagus is subject to diverticula corrosive inflammation, strictures and tumors as occur in other parts of the gastro-intestinal tract.

TRACTION DIVERTICULUM OF THE ESOPHAGUS

A diverticulum of the esophagus can occur anywhere along the esophagus and can be acquired, congenital, or post-traumatic. The traction diverticulum occurring in the mid-third of the esophagus is an example of the acquired type; it results from adhesions between the hilar or mediastinal lymph nodes and the wall of the esophagus. The wall of the esophagus is pulled laterally and upward into a tent-like formation as demonstrated with barium contrast media. The diverticulum becomes filled with food or barium during the act of swallowing but the diverticulum readily empties with no retention of food or barium to be regurgitated later. Consequently, there is no surgical indication for resection of this diverticulum unless it becomes distorted and larger to retain food and barium which the patient later regurgitates (see Chapter 19).

Technique.—Through a right anterior thoracotomy or right posterior-lateral thoracotomy incision, the right mediastinal pleura is opened and the azygos vein ligated and divided so that the esophagus can be mobilized. In the mid-portion of the esophagus on the inner wall, the diverticulum will be found adherent to the bronchial or carinal lymph nodes. If the adhesion is cut without perforating the esophagus, the tent-like diverticulum retracts into the wall of the esophagus. It is evaginated with a blunt instrument into the main lumen of the esophagus and the muscular walls approximated with silk sutures. The mediastinal opening is closed with several interrupted silk sutures and the thoracotomy incision closed in the usual manner.

PULSION DIVERTICULA

Pulsion diverticula are probably congenital in origin and occur above a muscular sphincter or an area of narrowing of the esophageal lumen. Pulsion diverticulum is seen most commonly in the neck and then above the diaphragm; the latter has been called epiphrenic diverticulum and is situated above the diaphragmatic pinchcock. The cervical pulsion diverticulum occurs above the cricopharyngeus pinchcock. In both cases there must be a weakness in the muscles which allows the diverticulum to balloon out with the muscosal lining and produce a round sac.

TUMORS AND CYSTS OF THE DIAPHRAGM

Isolated benign tumors or cysts of the diaphragm should be resected and the defect in the diaphragm repaired with tantalum mesh or fascia lata. The lateral thoracotomy incision is best for this type of surgery.

EVENTRATION OF THE DIAPHRAGM

If the diaphragm becomes stretched and thinned out its effective motion for respiration becomes inadequate. The diaphragm becomes elevated and permits the abdominal viscera to move up into the thoracic cavity. This may lead to bronchial kinking and pulmonary atelectasis. The ineffectual diaphragmatic motion leads to a weak cough and permits secretions to collect in the dependent bronchus and results in a chronic pneumonitis. Abnormal vago-vagal reflexes have been described when the viscera slides up into the thoracic cavity. In such cases, surgery for the eventration must be done.

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As the sac becomes larger, it progresses downward and parallel to the esophagus. During deglutition of food or barium, it is seen to fill first and then the food overflows down into the esophageal lumen. Consequently, there is a constant retention of food and liquids in the sac and this leads to symptoms of regurgitation of food, fullness of the neck, and chronic cough due to laryngeal aspiration of regurgitated food. If the sac becomes distended with food and swallowed air, it may obstruct the adjacent esophagus and cause weight loss.

A pulsion diverticulum may develop following surgical resection of the portion of the muscular wall which was the site of a benign or malignant tumor. The mucosa bulges through the muscular defect of the esophagus and forms a round sac; it usually does not enlarge to the extent of the above described pulsion diverticulum. When symptoms of dysphagia or regurgitation of retained food from the sac or symptoms of chronic cough with intermittent attacks of asthmatic bronchitis due to tracheal aspiration occur, surgical treatment of the diverticulum is indicated. Surgical treatment consists of a one-stage or two-stage surgical procedure to remove the sac or to suspend it for adequate drainage and later surgical excision if indicated.

Technique: One-stage Procedure.—The diverticulum is exposed in the cervical region or thoracic region by the appropriate incision. The sac is dissected free at its origin from the esophagus. The stalk of the sac is clamped and the sac amputated leaving adequate mucosal cuff to make a two-row closure with continuous and interrupted sutures. The muscular layer is closed over with interrupted sutures. Levine tube drainage and feedings are continued for 5 to 7 days before oral feedings are started (*see* Chapter 10).

Technique: Two-stage Procedure.—Before the era of antibiotics this procedure was the safest and even with antibiotics, is the preferable procedure in some clinics, The diverticulum is exposed by the appropriate cervical or thoracic incision. The muscle layers covering the sac are dissected away from the mucosal lining of the sac. The sac is then sutured high in the cervical region or along the esophagus or other mediastinal structures so that it has dependent drainage at all times. At some later date, the surgeon can reopen the incision and dissect out the sac, amputating it at its origin from the esophagus. A careful two-row closure of the esophageal mucosa and the muscular layers is made. Levine tube drainage and feedings are carried on for 5 to 7 days before oral feedings are tried. The advantage of the two-stage procedure is that if leakage occurs, it will be localized and easily drained. In the one-stage procedure, leakage may lead to a diffuse mediastinitis.

PEPTIC ESOPHAGITIS

Peptic esophagitis is a complication of some hiatal herniations of the stomach or hyperacidity with regurgitation into the distal esophagus. The condition is very distressing to the patient with symptoms of substernal pain or burning sensation. Dysphagia is also due to the polypoid thickening of the mucosa and narrowing of the esophageal lumen. Medical management with antispasmotics and antacids relieves the acute symptoms. Surgical correction of the diaphragmatic hernia or vagotomy with gastro-enterostomy will often cure the condition More radical surgical procedures such as resection of the fundus and lower esophagus has been advocated.

PERFORATION OF THE ESOPHAGUS

Perforation of the esophagus may occur by trauma or spontaneously. Traumatic rupture or perforation is most common and usually due to a foreign body or instrumentation. Stab and gunshot wounds have been reported as a cause of esophageal perforation. Spontaneous rupture or perforation of the esophagus probably never occurs in a normal esophagus. Esophageal ulceration in the lower third predisposes to the perforation especially if violent vomiting or retching occurs. Any perforation of the esophagus is associated with early surgical emphysema in the supra-clavicular fossæ and diffuse mediastinitis. The toxicity associated with the latter is severe and requires maximum antibiotic and sulfa therapy. Perforations of the lower esophagus are associated with regurgitation from the stomach. Gastric secretions quickly erode through the mediastinal pleura and lead to a highly irritating pleuritis with effusion. Pneumothorax results from swallowed air passing through the perforation into the pleural cavity. The toxicity of the mediastinitis is greatly increased by the toxic pleuritis and empyema.

Treatment is medical or surgical. Medical management is instituted for the small perforations and high perforations and consists in constant Levine tube drainage and feedings from 5 to 7 days before oral feedings are permitted. Surgical treatment consists of thoracotomy with closure of the esophageal perforation. This also permits evacuation of the empyema and closed water seal drainage to re-expand the lung.

SHORT ESOPHAGUS

Short esophagus is congenital or acquired. There is some question as to the congenital type because of its rarity. The majority of cases are acquired and are the result of esophagitis associated with reflux as in diaphragmatic hernia or corrosive esophagitis with severe damage to the mucosa and muscularis. The resulting fibrosis of the esophageal wall leads to longitudinal shortening of the esophagus and this pulls the stomach up into the thoracic cavity. Barium studies at first are deceiving as the stomach presents as a large ampulla of the lower esophagus. In time, the stomach can be definitely visualized and the esophageal lumen is narrowed in the area of fibrosis.

Major thoracic surgery has little to offer because if the stomach is pulled down, it is gradually pulled back up into the chest. Transplantation of the esophagus to the central elevated portion of the diaphragm has been done with some success. Temporary or permanent crushing of the phrenic nerve can be tried first and the permanent exeresis done later when symptoms recur and phrenic nerve function returns.

STRICTURE OF THE ESOPHAGUS

Stricture of the esophagus may be due to congenital webs of the esophagus as reported in the newborn. The narrowing of the esophageal lumen leads to some difficulty in deglutition of coarse foods but not to semisolid foods. Acquired strictures are more common and result from severe reflux esophagitis or corrosive esophagitis. They may be single or multiple and of all sizes so that the esophageal lumen is compromised and will only allow the passage of clear fluids. Corrosive strictures are usually in the upper and lower thirds of the esophagus more often in the lower third. Reflux esophagitis leading to stricture formation, as well as

short esophagus, is located in the distal third. The stricture is clinically significant if the patient is unable to maintain his weight and continues to lose weight. The portion of esophagus above the stricture often dilates and retains food for some time before it can pass through the area of stricture. Such retained foods may be vomited easily or regurgitated into the mouth if the patient leans over to pick something up. Also, during sleep, the retained food may be aspirated into the tracheobronchial passages to give rise to a chronic bronchitis with cough and expectoration.

Treatment is primary dilation of the strictured area with bougies. Surgical resection of strictured area with an end-to-end anastomosis of the esophagus has been reported. Surgical resection of the distal third of the esophagus containing the strictured area with esophago-gastric anastomosis in the sub-aortic region is sometimes done.

CARDIOSPASM

Cardiospasm is a condition similar to stenosis of the lower esophagus. In cardiospasm there is no actual damage to the esophageal mucosa but considerable thickening of the muscular layers at the esophago-gastric junction. The condition is similar to idiopathic pyloric obstruction in the newborn. Various theories as to the etiology have been proposed and the absence of ganglion cells of the myenteric plexus of the lower third of the esophagus is one theory. Differential diagnosis of fibrostenosis due to esophagitis and stenosing carcinoma of the lower third of the esophagus must be considered before therapy. Treatment has consisted of single or multiple dilations of the area of spasm. Dilation is reported to be successful in 85 per cent of the cases, single or multiple dilations carry a risk of esophageal perforation. Various surgical procedures have been used but the Heller operation is used widely today.

Technique.—A left anterior or lateral thoracotomy is done with trans-diaphragmatic exposure of the stomach, distal third of the esophagus and esophago-gastric junction. A small gastrostomy opening is made high on the anterior wall of the stomach. The left index finger is inserted into the stomach and then into the lumen of the lower esophagus. The esophageal and gastric muscularis is incised carefully for a distance of 3 to 5 inches above and below the esophago-gastric junction. Care is used to avoid incision or perforation of the esophageal or gastric mucosa. After withdrawing the left index finger from the gastrostomy opening, it is seen that the esophageal and gastric mucosa herniate through the area of divided muscularis of the esophagus and stomach. If any minute perforation of the mucosa occurs it is closed carefully with interrupted fine silk sutures. The area of cardiospasm due to the thickened esophageal muscle is no longer present and the esophageal lumen is unobstructed. The gastrostomy opening is closed. The diaphragm is closed anatomically and securely with interrupted silk sutures. The thoracotomy incision is closed in the usual manner. Follow-up barium studies of the esophagus show gradual shrinkage of the dilated and tortuous esophagus back to normal with absence of the area of obstruction.

CARCINOMA OF THE ESOPHAGUS

Carcinoma of the esophagus is the most serious carcinoma of the gastro-intestinal tract. Complete obstruction occurs quickly and cannot be palliatively by-passed.

The clinical signs of weight loss and dysphagia are severe and progressive. The highest incidence of carcinoma of the esophagus occurs in the sixth and seventh decades and surgical excision of the carcinoma should be considered in all cases. Because of the magnitude of the surgery involved, careful preoperative evaluation is very important. Microscopic diagnosis of the tumor by endoscopic examination and biopsy and barium radiographic studies will establish the diagnosis and the location of the lesion in 90 per cent of the cases.

Technique.—The abdomen is always opened first through a left paramedian incision from the xiphoid to a level below and lateral to the umbilicus. If liver metastases or lymph node metastases about the celiac axis and superior edge of the pancreas exist, the lesion may be resectible but not likely curative. Frozen section of any gland at the esophago-gastric junction will determine if the primary tumor has extended beyond the thoracic limits. We have never found the thoracic lesion inoperable if the abdomen is free of metastases. The entire stomach is mobilized by ligature and division of the left gastric, left gastro-epiploic and the short gastric arteries. Minimal trauma of the stomach is essential during its mobilization. The spleen is rarely removed. The suspensory ligament of the left lobe of the liver is cut for exposure to the esophagus. The lower abdominal and diaphragmatic portions of the esophagus are mobilized about 6 inches distal to the ligament of Tritz, and a size 10 Levine tube is passed through a small stab wound of the jejunal wall and retrograde into the stomach. The tube is sutured to the jejunal wall with interrupted silk sutures and brought out of the abdominal cavity through a stab wound in the left upper quadrant. Constant suction is maintained on this tube with frequent small irrigations to be sure the lumen is open. Distal to the Levine tube a small catheter is inserted into the jejunum for a short distance and anchored to the jejunal wall with interrupted silk sutures. The catheter end is brought out through a stab wound in the left upper quadrant to be used as a feeding tube later on. Both tubes are well anchored to the skin by wire sutures. The abdomen is then closed with interrupted wire sutures and the right anterior thoracotomy incision is made in the fourth interspace. After the pleural cavity is opened and the lung mobilized, the azygos vein is doubly ligated and divided. The mediastinal pleura is opened upward and downward posterior to the right hilum. The entire esophagus and tumor is mobilized down to the diaphragmatic portion of the esophagus. The stomach is then pulled up through the normal hiatus into the right chest without twisting it. The esophagus is amputated at the esophago-gastric junction with ligation of the esophageal stump and a purse-string closure, as well as interrupted silk suture closure of the esophageal stump. The greater curvature of the stomach is then sutured to the anterior mediastinal pleura with multiple sutures so that the upper end of the stomach cannot slide down. The stomach is usually anchored to the apex of the right chest. The upper end of the esophagus is amputated high where it emerges from the upper mediastinum. A new incisional opening is made in the stomach and an end-to-side esophago-gastrostomy anastomosis is done. The anastomosis is folded in by rolling the stomach into a tube and again suturing it to the anterior mediastinal pleura. The right chest incision is closed in the usual manner.

If the esophageal tumor is very high, it may be necessary to bring the stomach out of a right supra-clavicular incision and amputate the esophagus in the lower cervical region. The regular anastomosis is then done in the supra-clavicular fossa.

Early ambulation of the patients is carried out except for the intermittent suc-

in the aspirating bottles would be desirable for the postoperative care. The danger of overloading the cardiovascular system with resultant heart failure is much greater in the case of pneumonectomy or the patient with moderate pulmonary emphysema and early cor pulmonale. On the other hand, the patient in chronic shock with a blood pressure below 80, suffers from circulatory hypoxia and anoxia. The combination of respiratory and circulatory hypoxia and anoxia is very destructive and many manifest itself by sudden death or start an irreversible fatal course.

As the surgeon is closing the thoracotomy incision, his postoperative treatment for overcoming any hypoxia or anoxia should be formulated and started. A prolonged period of shock during surgery should caution the surgeon in the use of fluids and blood for hypotension. To keep down the intravenous fluid volume, vascular stimulants (neosynephrin, levophed, etc.) may be given slowly by intravenous drip (5 per cent glucose in water) in concentrated amounts to maintain the blood pressure at a desired level.

It usually requires the first twenty-four hour postoperative period to stabilize the blood pressure within normal limits and to be certain that respiration is adequate. The first urine specimen is obtained during this period and the specific gravity is determined. A low specific gravity could indicate some renal impairment and it is not advisable to push fluids either by mouth or intravenously until later urine specimens show a specific gravity over 1.012–1.015. Minimal opiates are given for pain during this period for fear of depressing respiration. Antibiotics are started

SECOND TWENTY-FOUR HOUR PERIOD

At the end of the first twenty-four hour period, blood pressure should be over 100, respiration 24 to 28, and pulse between 80 to 100 per minute. The red blood count and hemoglobin should be within normal limits. The lungs should be relatively clear on auscultation and a portable chest *x*-ray should show the contralateral lung clear and the operated lung absent (pneumonectomy) or expanded (lobectomy or exploratory). The temperature may vary from subnormal to 101 degrees or more. Urinary output will be diminished during this period and the possibility of oliguria or anuria cannot be ascertained this early. The patient is started on oral liquids at this time and all intravenous fluids stopped. Hypodermic opiates are stopped and oral analgesics are prescribed. The patient is encouraged to cough frequently to maintain clear air passages. The patient is permitted to sit up in bed and oxygen is switched from a tent to a nasal tip catheter to permit freedom of movement in bed.

THIRD TWENTY-FOUR HOUR PERIOD

By this time, the blood pressure, pulse and respiration should be the same as before surgery. Urinary function can be determined from the output and if it is increasing, liquids and food can be given freely. The temperature may be over 100 degrees and the red blood count and hemoglobin may show secondary anemia due to the increased hydration. If the secondary anemia is 50 per cent of normal, small daily blood transfusions may be indicated.

Atelectasis due to bronchial obstruction or to compression by fluid or blood in the chest cavity must be relieved. Wheezing or emphysematous breathing due to bronchiolar obstruction must be relieved by bronchial dilator drugs. The patient

sits up on the bed side and dangles his feet three times a day for five minutes. If water-seal tubes are present, usually the nonfunctioning upper tube can be removed.

FOURTH TWENTY-FOUR HOUR PERIOD

By this time the patient should be on a regular diet, sitting in a chair three times a day for ten minutes. All water-seal tubes are removed. Any persistent temperature over 100 degrees indicates likely pneumonitis of the lung or infection of the pleural cavity as an impending empyema. For a suspect pneumonitis, expectorants and increased antibiotics are prescribed with encouragement to cough. An impending empyema requires aspiration of any fluid or blood in the chest cavity with instillation of antibiotics. Tachycardia is usually secondary to obscure infection, azotemia, anemia, or myocarditis. Any cardiac arrhythmia should be checked by electrocardiogram and the patient should be started on digitalis preparations and quinidine. When the pulse rate becomes regular, quinidine is discontinued and digitalis preparation continued for some time. Cardiac arrhythmia probably indicated some hypoxia and intermittent oxygen therapy is continued for several weeks postoperatively. Patients are allowed to wean themselves of oxygen and encouraged to use it during the sleeping hours.

By the fifth postoperative day, the patient may go to the bathroom and thereafter assume as much activity as he desires. Antibiotics are continued until the temperature has been normal for forty-eight to seventy-two hours and the white blood count is 15,000 or less. Stitches are removed on the sixth postoperative day and hospital discharge usually is on the tenth to fourteenth postoperative day.

COMPLICATIONS

Unusual complications can occur after any surgery but the following are seen during the postoperative period of thoracic surgery.

Blood Transfusion Reaction.—Because of the blood transfusions given during surgery to maintain the vascular tension within normal limits, reactions to the transfusion may vary from allergic urticaria to extreme pain in the kidney and lumbar areas. The mild reaction will respond to antihistamine or cortisone therapy. The more severe reactions associated with jaundice or hemoglobinuria may have oliguria for several days or become anuric.

The use of an artificial kidney must be considered, but therapy is started of limited fluids by intravenous and oral routes and daily blood chemistry for electrolyte balance. Hypertension develops as the azotemia increases. ACTH or cortisone therapy is indicated. When diuresis occurs careful electrolyte balance of the blood must be maintained.

Persistent Atelectasis.—Following lobectomy or segmental lobectomy, persistent atelectasis of a lobe or segment of a lobe may persist. Cause for same may be: failure of patient to cough because of chest pain, thick tenacious secretions which fail to come up on coughing and operative or traumatic edema of the bronchial mucosa.

Patients vary in their threshold for pain and it may be necessary to do procaine intercostal blocks of the operated chest so that the patient is relieved of pain and can effectively cough. Otherwise, passing a catheter through the nose into the posterior pharynx and larynx will stimulate forceful coughing. If this fails to dislodge the accumulated secretions, bronchoscopy can be done with minimal topical

anesthesia. Early use of oral expectorants helps to prevent bronchial secretion from becoming so tenacious.

Persistent Hemothorax.—Following a thoracic surgical procedure where the lung was adherent to the chest wall, persistent hemothorax or serosanguineous fluid may be troublesome in spite of adequate tube drainage or adequate chest suction. At the time of surgical closure, the chest cavity may have appeared dry of any bleeding but postoperative oozing of the chest wall occurs.

Irrigation of the tubes with a penicillin solution (1000 units per ml.) may improve the drainage of serosanguineous fluid. If breath sounds are heard on auscultation in spite of a chest roentgenogram showing complete opacity of the chest cavity, the situation is not too serious. Breath sounds on auscultation are more significant than the radiographic findings, and the former should be elicited several times daily to indicate possible accumulation of bronchial secretions within the partially obstructed bronchi. A good cough reflex is essential and respiratory exercises such as deep breathing or blowing up balloons increases the tube drainage or absorption of the fluid. If the chest radiographic opacity persists with auscultatory findings of absent to diminishing breath sounds, re-operation of the chest may be considered for removal of the hematoma and decortication and re-expansion of the underlying lung.

Pleural Effusion and Empyema.—Pleural effusions are secondary to postoperative or post-traumatic chest complications, cardiac, renal or hepatic insufficiency and neoplastic disease of the pleura or lung or pulmonary infections which extend through the visceral pleura into the pleural space.

Any benign pleural effusion may become infected from infection within the lung, from postoperative bronchial leak or pulmonary fistula, from outside of the chest wall (as by contaminated needle aspiration) or retrograde through the diaphragm from the peritoneal cavity (peritonitis). All tests of the pleural fluid such as specific gravity, bacteriology, organic and inorganic chemistry and microscopic studies may fail to indicate when a benign fluid has become an empyema. However, elevated body temperature, purulent fluid and thickened parietal pleura at the time of needle aspiration usually indicates an empyema. Repeated daily aspiration of the fluid with instillation of large amounts of the specific antibiotics may clear the pleural infection. Systemic antibiotics must be maintained if the patient also has a primary pulmonary infection as the cause of the empyema.

If the empyema is associated with cough and expectoration and the expectoration is the same as obtained by needle aspiration, a bronchopleural fistula exists. A chest roentgenogram showing an air-fluid level in the chest in which the air has not been injected or aspirated into the chest during the fluid aspiration, indicates a bronchopleural fistula. This represents an emergency as the patient will continue to cough until all the empyema fluid is evacuated and the space is filled with air. During the coughing, a bronchogenic spread of the empyema fluid throughout both lungs with severe bilateral pneumonitis may occur. Water-seal drainage must be instituted as soon as the diagnosis is established using a large de Pezzer catheter. If there is any question about the diagnosis, 2 centimeters of sterile methylene blue may be injected into the empyema space and if the patient coughs up the dye, the diagnosis is obvious. Also, a sample of the air in the chest may be obtained and analyzed for oxygen and carbon dioxide. If the results simulate the oxygen and carbon dioxide concentration of air, the diagnosis is a bronchopleural fistula.

After water-seal drainage has been established, there will be bubbling through

the water-seal setup with drainage of purulent secretion from the pleural cavity. The cough and expectoration are much relieved. In infants and young children, closed water-seal drainage is instituted as soon as the diagnosis is established to eliminate toxic absorption manifested by high temperature and convulsions.

SURGICAL EMPYEMA AND LUNG ABSCESS

When an empyema has failed to clear after multiple needle-syringe aspirations and instillation of antibiotics into the empyema space and the pleura has become thickened with purulent exudate, open thoracic surgical drainage is instituted.

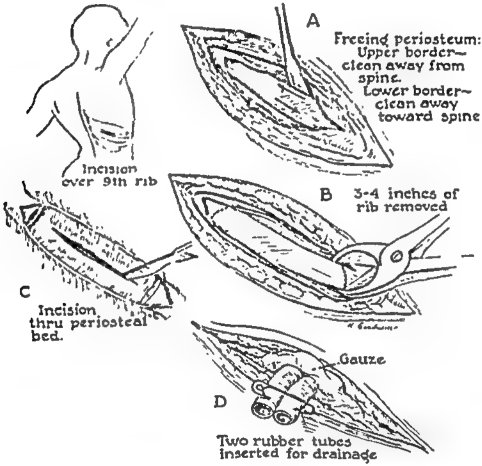


Fig. 228.—Open method of drainage.

The empyema is chronic at this stage and well localized; there is no fear of opening into a free pleural cavity with a resulting open pneumothorax. If the chronic empyema is complicated by a bronchopleural fistula, immediate open thoracic surgical drainage is indicated or closed water seal drainage must be instituted using a large de Pezzer catheter until the infection becomes well localized.

Technique.—Open drainage of an empyema is usually done under local anesthesia. If a bronchopleural fistula also exists, the patient must be in a sitting or semi-sitting position or the empyema will drain through the bronchopleural fistula into the contralateral lung. Hence, the importance of doing the procedure under local anesthesia so that the patient can expectorate all tracheobronchial accumulation. The empyema space is aspirated in its most dependent portion using needle aspiration under local anesthesia. The skin area over the rib superior to the de-

pendent portion of the empyema is blocked with 1 per cent procaine—25 ml intracutaneously and subcutaneously. A 4- to 6-inch incision is made over the rib and all bleeding controlled. The muscle layers over the rib are blocked (with 1 per cent procaine—10 ml. and divided. The ribs of the chest wall are exposed and the intercostal space above and below the selected rib for resection is infiltrated with 1 per cent procaine until the tissue is swollen and edematous. Subperiosteal resection of the rib is done and usually a segment of 4 to 6 inches of rib is removed. The subperiosteal bed and thickened parietal pleura are incised enough to aspirate the purulent contents of the empyema and to insert a finger and palpate the bottom of the empyema space. The rib below or above is resected in the same manner to get to the bottom of the empyema space.

The intervening intercostal muscle bundle and intercostal vessels and nerve are ligated and divided at the cut rib ends. A circular segment of the parietal pleura is resected so that an opening of 2 to 4 inches is obtained. The skin edges are sutured to the upper and lower edges of the thickened parietal pleura. In this way all exposed edges of muscle and rib are covered by the skin edges and a bronchopleurocutaneous or pleurocutaneous fistula is made. The wound is permitted to drain into large absorbent dressings and changed as indicated. The patient is encouraged to lie on the operated side to obtain dependent drainage, and early ambulation is started with maximum activity to stimulate drainage and motion of the incarcerated lung and diaphragm. Healing occurs slowly with gradual sloughing of the thick exudate on the visceral and parietal pleura and over the surface of the diaphragm. The bronchopleural fistula gets smaller and finally closes with no further evidence of fistulous wheezing or whistling on deep respiration.

Gradually motion is seen in the underlying lung and diaphragm on coughing and deep breathing. The lung re-expands to fill the empyema space and becomes adherent to the parietal pleura. The diaphragmatic motion returns, and the pleurocutaneous opening gets smaller until only a small fistula persists and finally heals leaving a depressed scar on the chest wall. After several years, a minimal amount of paradoxical motion may be seen in the area of the resected ribs on deep respiration and plastic repair of the scar may be considered.

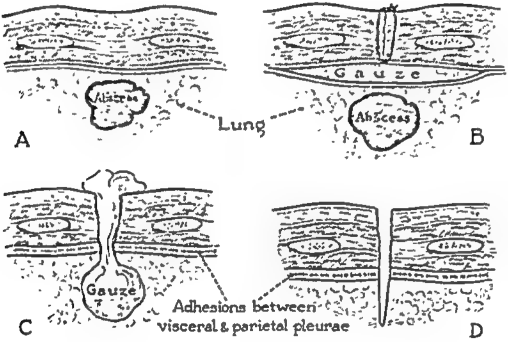
Lung Abscess.—Lung abscess is symptomatically the same as an empyema with bronchopleural fistula. Roentgenograms are often similar. Rationale of therapy is the same as for empyema with bronchopleural fistula and open thoracic surgical drainage may be considered. Because of the possibility of a free pleural space or minimal obliteration of the pleural space in the area of the pulmonary abscess, a two-stage drainage procedure is used.

Technique.—The lung abscess is localized by *x*-ray and fluoroscopic study and after careful subperiosteal resection of the segment of rib or ribs over the abscess is carried out under local anesthesia, the subperiosteal bed is packed with iodoform gauze and sutured loosely in place before the chest wall muscles and skin edges are closed with interrupted silk sutures.

Seven to ten days later, under local anesthesia, the wound is reopened and the iodoform gauze pack removed. Needle aspiration of the abscess cavity is done through the subperiosteal bed and through the area of foreign-body reaction and obliterated pleural cavity. When the lung abscess has been localized, the peripheral wall of the abscess is incised and the intervening intercostal muscle bundle and intercostal vessels and nerve are ligated and resected. A round segment of the parietal pleura is resected with ligation of all bleeding points; then the skin edges

are sutured to the pleural edges with interrupted silk sutures. Thus, a broncho-pulmonary-pleuro-cutaneous fistula is produced. Healing occurs by gradual closure of the pulmonary and bronchial fistulæ until no wheezing is heard or air can be expelled through the opening on forced expiration. Then the abscess cavity becomes smaller as well as the drainage opening. Finally a small draining fistula persists for a few days to a few weeks and then heals completely. The chest roentgenogram shows a fibrotic scar in the area of the previous abscess.

The above healing process of a lung abscess is associated with residual bronchiectasis in the area of the healed abscess, and the bronchiectasis may be the source of continued hemoptysis and expectoration. To circumvent the residual bronchiectatic complication, a lobectomy is often done to remove the lung abscess.



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Pleural effusion, empyema, empyema with bronchopleural fistula and lung abscesses are secondary diseases and while treatment is being carried out, the primary disease must be ascertained. Often the treatment of the secondary disease may be modified or changed completely from the foregoing. For example, lung abscess distal to a bronchial tumor can be treated best by lobectomy or pneumonectomy to eradicate the abscess and the tumor.

QUESTIONNAIRE

1. Which is the most crucial postoperative period?
2. What precaution must be used after the endotracheal tube is removed following pneumonectomy?
3. When should the decision to remove the endotracheal tube be made? Explain.
4. How is oxygen given?
5. Discuss the importance of blood volume replacement.
6. Discuss the danger of overloading the cardiovascular system.
7. How is the blood pressure maintained postoperatively?
8. What does a urine with low specific gravity indicate?

9. Why should only minimal opiates be administered?
10. What should the blood pressure, respiratory rate and the pulse be at the end of 24 hours, postoperatively?
11. Discuss the plan for postoperative care during the succeeding five days.
12. Discuss the blood transfusion reactions that may occur.
13. What are the causes for a persistent atelectasis?
14. What is the treatment for the above?
15. Give the treatment for postoperative persistent hemothorax.
16. When is re-opening of the chest wall considered for the above?
17. To what is pleural effusion secondary?
18. How may a benign pleural effusion become infected?
19. What are the signs and symptoms indicating empyema?
20. What is the treatment for pleural infection?
21. What indicates the existence of a bronchopleural fistula?
22. What is the treatment for the above?
23. When is an open thoracic surgical drainage indicated for an empyema?
24. Describe the technique for the open drainage method.
25. Discuss the management of postoperative lung abscess.

CHAPTER 41

THE ABDOMINAL WALL

By A. V. Partipilo

"It is not merely by holding a knife in a certain position, or making an incision of a definite length or form on the surface of the body, that an operation is performed; unless a person has a competent familiarity with the structures under the surface, he will do well not to interfere with cutting operations."—Sir William Fergusson, 1842.

GENERAL CONSIDERATIONS

The Abdominal Cavity, unlike the thoracic cavity, is enveloped anteriorly and laterally by a group of muscles and fasciæ. This arrangement is necessary in order that the abdominal wall may aid in the process of respiration. A flexible abdominal wall is also essential to overcome an increase in the volume of the abdominal cavity. A normal increase occurs after a hearty meal, when the stomach may enlarge two or more times its size. This increased volume is compensated by a corresponding relaxation of the abdominal wall.

In abdominal disease the wall will expand or contract, depending upon the nature of the disease. For instance, in intestinal obstruction the intra-intestinal pressure is tremendous, and nature compensates by distending the abdominal wall. On the other hand, in peritonitis the abdominal wall contracts and becomes rigid, thereby decreasing the intra-abdominal volume. In both instances the response of the abdominal wall is protective, in the former instance allowing greater area for the increasing intestinal pressure, and in the latter instance diminishing the area, thereby limiting intestinal movement in an effort to localize the infection. The subject of abdominal pressure is very important to the surgeon; a supplementary study should be made.

Body Habitus.—The contour of the abdomen varies according to the obesity of the patient. If obese, the abdomen is smooth, round, and protuberant. In thin subjects it is depressed, and the margins of the thorax, the iliac crests, and the pubis stand out. The configuration of the abdominal wall also varies with age, sex, muscular development, and the presence of tumors.

The shape of the body and the position of various abdominal organs vary within wide limitations. Four types of body habitus are recognized:

1. *Hypersthenic:* these are the massive, powerful individuals having the following characteristics:
   - (a) Heavy bony framework.
   - (b) The thorax is wide and deep; short from above downward; the heart is transverse in position.
   - (c) The subcostal angle is wide and the xiphoid-sternum is broad and large.
   - (d) The stomach and intestines are high in position.
   - (e) The colon is high and the transverse colon is transverse in position.
   - (f) About 5 per cent of individuals are of this type.

2. *Sthenic Type:*
   (*a*) About 48 per cent of individuals constitute this type.
   (*b*) Their characteristics are similar to but not as exaggerated as the hypersthenic type.
   (*c*) Most stout men fall under this group.
3. *Asthenic Type:*
   (*a*) About 12 per cent of individuals constitute this group.
   (*b*) Bony-framework is light and they are the slender and frail type.
   (*c*) Thorax is long and narrow.
   (*d*) Subcostal angle is very narrow and the xiphoid-sternum is also slender.
   (*e*) The characteristic feature of this type is the low position of the gastrointestinal tract; the pylorus may be found within the pelvis.
   (*f*) The transverse colon drops low into the pelvis.
4. *Hypoasthenic Type:*
   (*a*) About 35 per cent of individuals fall into this group. Their characteristics are similar to the asthenic except that they are less pronounced.

TOPOGRAPHICAL ANATOMY

Linea Alba.—A depression is noted in the mid-plane extending from the tip of the sternum to the pubis. This is called the linea alba, commonly designated as the "white line." It is formed by the fusion of the fasciæ of the abdomen. On either side of this line are found the recti muscles.

The linea alba is of surgical importance because it is the only part of the abdominal wall which is devoid of muscles and has a limited blood supply. For this reason it is the region through which the surgeon chooses to introduce a trocar to tap the peritoneal cavity.

Linea Semilunaris.—The linea semilunaris is seen in well-developed subjects on either side of the recti muscles. It corresponds to the aponeurotic sheath of the internal oblique, as it splits to surround the recti muscles. It is also known as Spigelian line.

The linea semilunaris is an important surgical landmark. It is the site which the surgeon uses at times for making incisions. It should be emphasized, however, that incisions made through this area will sever one or more of the nerves supplying the rectus muscle. The inevitable result will be a partial paralysis with subsequent weakening of the abdominal wall. The fascia is not overlapped by muscle; hence reconstruction of an incision is imperfect. These factors favor the development of postoperative hernia. It must also be borne in mind, when making an incision below the umbilicus, that the inferior epigastric artery crosses the linea semilunaris.

Surface Markings.—A knowledge of the abdominal surface markings and the relationship of the peritoneum with the various abdominal organs is essential for diagnosis and treatment. The surgeon must be able to visualize clearly the exact location of abnormal and normal structures in the abdomen. The following are the fixed landmarks of the abdomen:

1. Right and left anterior superior spines of the ilium.
2. Right and left tubercles of the crest of the ilium; found two to three inches posterior to the anterior superior spine.
3. Symphysis pubis.

4. Pubic tubercles; about 1 inch lateral to the symphysis pubis. Just lateral to the tubercles are found the spermatic cords.
5. Tip of the xiphoid process of the sternum.
6. Lower border of the tenth rib.
7. Umbilicus; a variable landmark.

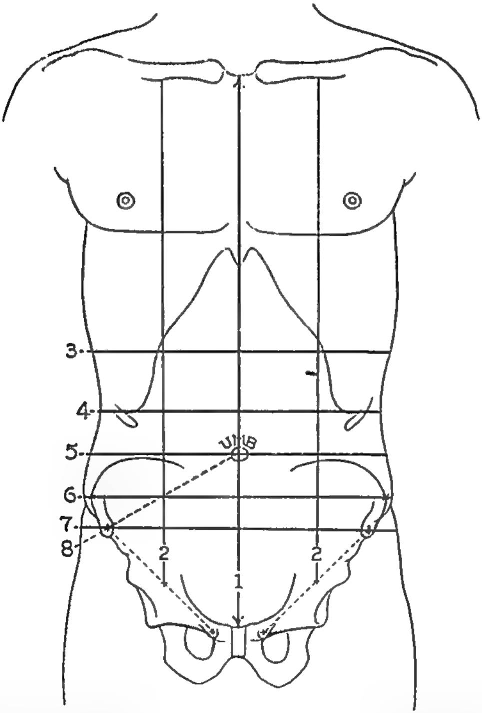

FIG 230 —Lines and planes of the abdomen: *1*, median vertical; *2*, lateral vertical; *3*, transpyloric plane; *4*, subcostal plane; *5*, umbilical; *6* intertubercular line; *7*, interspinous line; *8*, spino-umbilical line.

Rules in Abdominal Topography.—1. *Topographical Point.*—(*a*) A point bisected by two straight lines; example, the gall-bladder point is located at the junction of the outer border of the right rectus to the costal margin.

(*b*) A point on a line between two landmarks, example, McBurney's point is found 2 inches medial to the anterior superior spine on a line drawn from that process to the umbilicus.

(*c*) A bony landmark is a fixed point.

2. *Topographical Line.*—(*a*) The shortest distance between two topographical points or bony landmarks; example, the intertubercular line extends between the two iliac tubercles.

(*b*) A line drawn either horizontally or vertically to a fixed point; example, the median vertical is made vertical to the symphysis pubis.

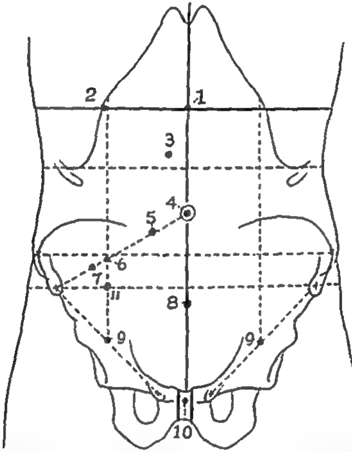

Fig. 231 —Abdominal points: *1*, Addison's point; *2*, gall bladder point; *3*, Robson's point; *4*, Umbilicus; *5*, Morris' point; *6*, Munro's point; *7*, McBurney's point; *8*, mid-hypogastric point; *9*, femoral point; *10*, symphysis; *11*, Lanz's point.

3. *Topographical Triangle.*—Formed by joining three topographical points or bony landmarks.

Lines and Planes of the Abdomen.—1. Transpyloric Plane.—Also called Addison's or Mid-epigastric Plane. It extends at a right angle to the median vertical plane at a point mid-way between the suprasternal notch and the symphysis pubis. This plane is of immense practical value since it crosses the first lumbar vertebra, the pyloric end of the stomach, and marks the course of the pancreas. It is also used by Addison as the pivotal plane for dividing the abdomen into zones (Fig. 230).

2. Subcostal Plane.—Passes beneath the tenth costal cartilage. This plane varies considerably; in some individuals it may reach the border of the crest of the ilium.

3. Intertubercular Plane.—Extends between the two iliac tubercles.

4. Interspinous Plane.—A line drawn between the anterior superior spines of the ilium.

5. Umbilical Plane.—Drawn at a right angle to the median vertical plane at the umbilicus. Due to the variable position of the umbilicus, this plane is very seldom used.

6. Spino-umbilical Line.—Drawn from the anterior superior spine to the umbilicus.

7. Median Vertical Plane.—Extends from the tip of the xiphoid process, or from the center of the suprasternal notch, to the symphysis pubis. On this line is located the linea alba

8. Lateral Vertical Plane.—A line drawn parallel with the median vertical at a point mid-way between the pubic tubercle and the anterior superior spine; corresponds to the location of the linea semilunaris.

Abdominal Points.—1. Addison's Point.—Located on the median vertical plane at a point halfway between the suprasternal notch and the symphysis pubis (Fig. 231).

2. Gall-bladder Point.—The point of the greatest tenderness in gall-bladder disease. It is located at the junction of the transpyloric plane with the right costal margin.

3. Robson's Point.—Located, according to Robson, "just above and to the right of the umbilicus," where tenderness on pressure exists with disease of the pancreas.

4. Umbilicus.—The contour and configuration of the abdomen must be taken into consideration when using the umbilicus as a landmark. It is often the location for ventral herniæ.

5. Morris' Point.—Located within 2 inches of the umbilicus on the spino-umbilical line. It is advocated as the point of tenderness when none is noted in McBurney's point.

6. Munro's Point.—Located at the junction of the spino-umbilical line and the right lateral vertical plane. It is the site of the ileocecal valve.

7. McBurney's Point.—Located 1½ to 2 inches medial to the anterior superior spine on a line drawn from that process to the umbilicus. Generally recognized as the point of tenderness in acute appendicitis.

8. Mid-hypogastric Point.—Located mid-way between the umbilicus and the symphysis pubis on the median vertical.

9. Femoral Point.—Also called the Mid-Poupart point. It is found mid-way on a line drawn from the pubic tubercle to the anterior superior spine.

10. Symphysis Pubis.

11. Lanz' Point.—Located at the junction of the right lateral vertical plane with the interspinous line. It is proposed as the site of tenderness in chronic appendicitis. Its value is doubtful.

Triangles of the Abdomen.—1. Hesselbach's Triangle.—Its base is formed by the line of the inguinal ligament, its medial border by the rectus abdominis muscle, and laterally by the inferior epigastric artery. It is the site through which direct herniæ occur.

2. Lumbar Triangle of Petit (Fig. 232).—Bounded in front by the external oblique, behind by the latissimus dorsi, and at its base by the crest of the ilium. Rarely, lumbar hernia occur in this triangle. It is the site through which a lumbar abscess may come to the surface.

3. Superior Lumbar Triangle (Fig. 233).—Its base is formed by the twelfth rib and the lower border of the serratus posticus inferior; in front by the posterior border of the internal oblique; and behind, by the anterior border of the erector spinæ. The triangle is covered by the latissimus dorsi.

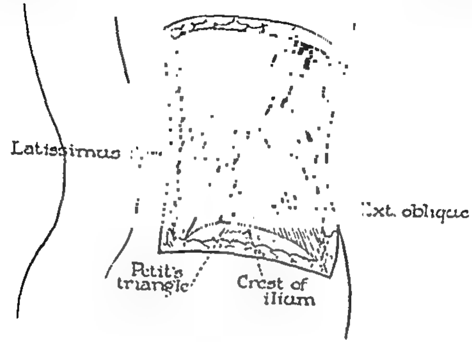

FIG. 232.—Lumbar triangle of Petit.

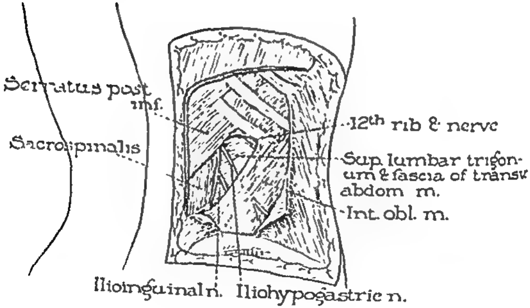

FIG. 233.—Superior lumbar triangle.

4. Femoral Triangle (Scarpa's or Subinguinal).—Base is formed by the inguinal ligament, the inner border by the adductor longus muscle, and the outer border by the sartorius muscle. It is the location of the femoral vessels and nerves and the site for femoral hernia.

Abdominal Zones.—Many systems have been devised for dividing the abdomen into zones for the purpose of localizing and determining the relationship of the various abdominal structures. Addison recognized only five of the many surface

markings on the anterior body wall as of value for dividing the abdomen into zones. These are:

1. The suprasternal notch.
2. Symphysis pubis.
3. Right anterior superior spine.
4. Left anterior superior spine.
5. The mid-point of the mid-line of the body (Addison's point).

Addison found, after careful measurements of 40 bodies, that these points are the most constant. He pointed out that the costal margins are unreliable due to the fact that they vary to a considerable extent in different individuals. In some cases the subcostal plane may coincide with the umbilical or even the intertubercular

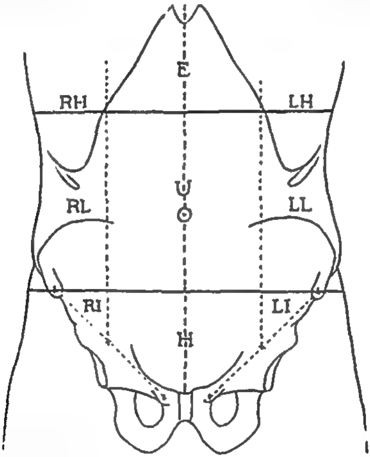

FIG 231 —Addison's abdominal zones: *RH*, right hypochondriac; *E*, epigastric; *LH*, left hypochondriac; *RL*, right lumbar; *U*, umbilical; *LL*, left lumbar; *RI*, right inguinal; *H*, hypogastric; *LI*, left inguinal.

line. On the other hand, he found that a point mid-way between the suprasternal notch and the symphysis is remarkably constant. This point corresponds with the level of the first lumbar vertebra, and marks the attachment of the transverse mesocolon to the posterior body wall.

A line drawn at a right angle to this point marks a plane around the body within which are located the following structures: pylorus, the root of the mesocolon, the first part of the duodenum, kidneys, gall-bladder, pancreas, spleen, and the stomach.

The second plane of Addison is one drawn between the anterior superior spines of the ilium. This marks the lowermost plane of the body and overlies the fifth lumbar vertebra.

As illustrated in Figure 234, when the two horizontal planes are crossed by the two lateral vertical lines, the abdomen is divided into nine zones. The value of Addison's topographical markings is that they include an area between the xiphoid-sternal notch and the transpyloric plane, within which are included many important organs and structures.

In Addison's abdominal topography the following zones are found: (1) epigastric; (2) right hypochondriac; (3) left hypochondriac; (4) right lumbar; (5) umbilical; (6) left lumbar; (7) right inguinal; (8) hypogastric; and (9) left inguinal. The author suggests that, if necessary, the lumbar and the umbilical regions be further divided by the subcostal plane. In this case the umbilical and the lumbar regions may be termed the supra- and infra-umbilical and lumbar zones.

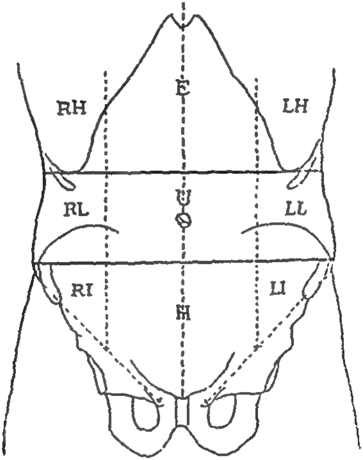

FIG. 235.—Abdominal zones using the subcostal plane and intertubercular planes.

Other systems include the subcostal, with the intertubercular line as the lowermost plane (Fig. 235). Some advocate a system of triangles, circles, or rhomboids. These are not practical and are often confusing. The author suggests Addison's method as the basis for all practical purposes. If it becomes necessary to localize further an abnormal structure or organ, other lines, angles, or triangles may be included or superimposed upon Addison's lines.

Muscles of the Abdominal Wall.—1. *Rectus Abdominis Muscle.*—This is a paired muscle located on either side of the linea alba. It is a broad, flat, strap-like muscle taking its origin from the symphysis. It is directed upward to become attached to the xiphoid process and to the anterior surfaces of the seventh, sixth, and fifth costal cartilages. On its surfaces are three or more tendinous intersections,

called inscriptiones tendineæ, which are firmly adherent to the anterior sheath of the rectus. The medial border of the muscle lies close to the linea alba, whereas, its lateral border corresponds to the linea semilunaris.

2. *Internal Oblique Muscle.*—Arises from the lateral half of the abdominal grooved surface of the inguinal ligament, from the anterior two-thirds of the iliac crest, and from the lumbar fascia. The direction of its fibers is from below upward and medially. *The insertion of the muscle is as follows:*

(a) Posterior fibers are attached to the cartilages of the last four ribs.

(b) Middle fibers are attached to the inferior borders of the seventh and eighth ribs, xiphoid process, and the linea alba.

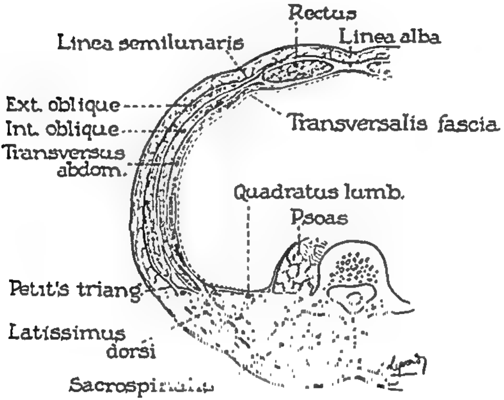

FIG. 236.—The lumbodorsal fascia and the origin of the fascia of the anterior abdominal wall.

(c) The lowermost fibers, of the inguinal ligament origin, join with the transverse abdominis muscle to form the conjoined tendon. The latter is inserted into the pubic crest and to the iliopectineal line.

3. *Transverse Abdominis Muscle.*—Has three points of origin:

(a) Pelvic origin—from the lateral one-third of the inguinal ligament, and the anterior two-thirds of the internal lip of the iliac crest.

(b) Costal origin—from the costal cartilages of the lower six ribs.

(c) Vertebral origin—through the medium of the lumbodorsal fascia it is attached to the spinous processes and the tips of the transverse processes of the lumbar vertebræ.

The muscle fibers are directed horizontally forward and end in an aponeurosis which has a two-fold insertion: (1) After taking part in the formation of the rectus sheath, it is attached to the linea alba, symphysis pubis, and the xiphoid process; (2) the fibers arising from the inguinal ligament join with the internal oblique

to form the conjoined tendon, to become inserted into the pubic tubercle and the iliopectineal line.

4. *External Oblique Muscle.*—Arises from the outer surfaces of the lower borders of the last eight ribs. From their point of origin the fibers proceed downward in various directions. The posterior fibers are directed vertically to become attached to the anterior half of the crest of the ilium. The middle and superior fibers end in a strong aponeurosis. Superiorly it is inserted into the xiphoid process, and inferiorly the aponeurosis folds upon itself (inguinal ligament) to become attached to the anterior superior spine of the ilium and to the pubic tubercle.

Fasciæ of the Abdominal Wall.—It will be necessary to study the lumbodorsal fascia in order to understand properly the fasciæ of the anterior abdominal wall.

The lumbodorsal fascia is composed of three layers which envelop the quadratus lumborum and the sacrospinalis muscle. Most authors consider the lumbodorsal fascia only that part which envelops the sacrospinalis muscle; however, we also include that part which forms the sheath for the quadratus lumborum.

The posterior sheath (superficial) is attached to the spinous processes of the lumbar vertebræ and to a part of the ilium and sacrum. The fascia covering the quadratus lumborum muscle arises from the anterior border of the transverse processes of the lumbar vertebræ. At the lateral border of the sacrospinalis the two sheaths of the muscle, with the thin fibers of the fascia of the quadratus lumborum, join to form the fascia transversalis, and give rise to the aponeurosis for the internal oblique muscle. From the posterior sheath a layer of fascia is derived for the latissimus dorsi and the external oblique (Fig. 236).

The transversalis is the thinnest of the three fasciæ of the anterior abdominal wall. Its fibers are thickest in the inguinal region, and become progressively thinner as the thorax is reached. The fascia lines the entire abdomen and is found between the abdominal muscles and their fascia on the one hand, and the peritoneum on the other. In the iliac fossa the fascia becomes continuous with the fascial lining of the iliacus and the psoas muscle. This part of the transversalis is called the fascia iliaca. In this area it is attached to the internal lip of the crest of the ilium. Superiorly it becomes continuous with the diaphragmatic fascia. In the inguinal region it is somewhat complicated and plays an important part in the posterior wall of the inguinal canal. This part of the fascia will be studied in *detail in the* chapter on herniæ.

The fibers run across the abdomen beneath the transverse abdominis muscle, and after forming the lowermost layer of the rectus sheath they end in the linea alba. From this point the fascia becomes continuous with the fascia transversalis of the other side.

Rectus Sheath.—The manner in which the rectus sheath is formed deserves some consideration. Above the subcostal plane and below the mid-hypogastric point, the posterior surface of the rectus muscle is covered only by the thin transversalis fascia (Fig. 238). However, anterior to the muscle is a strong fascia formed by the fusion of the fasciæ of the internal and external oblique muscles. At the mid-hypogastric point, where the rectus muscle rests upon the transversalis only, a sharply lunated fold—the semilunar fold of Douglas—is formed. The inferior deep epigastric artery enters the sheath of the rectus through this fold.

Because of the greater strength of the rectus sheath in the subumbilical region, and because the recti muscles are closely approximated in the mid-line, the gynecologist *often prefers the median incision for pelvic surgery.*

In the area of the abdominal wall between the mid-hypogastric point and the subcostal plane, the rectus muscle is covered both anteriorly and posteriorly by an equal distribution of fibers from the internal and external oblique fasciæ (Fig. 237). Just lateral to the rectus muscle the sheath is fused together and becomes directly continuous with the flat muscles. The point of fusion is called the linea semilunaris. Incisions in the linea semilunaris are unsatisfactory because reconstruction is imperfect. It is also impossible to preserve the nerve supply to the rectus muscle, when such incisions are made.

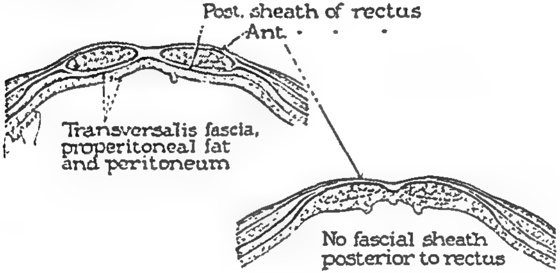

Fig. 237 Fig. 238

Fig. 237.—The rectus fascia as found between the subcostal plane and the mid-hypogastric point.

Fig. 238.—The rectus fascia above the subcostal plane and below the mid-hypogastric point.

Arteries of the Abdominal Wall.—1. *Inferior Deep Epigastric Artery.*—The inferior deep epigastric artery arises from the external iliac artery about one-fourth inch above the inguinal ligament, near the mid-femoral point. Its direction is upward and medially toward the mid-hypogastric point, where it enters the rectus sheath through the semilunar fold of Douglas; thence it proceeds superiorly, sending numerous branches to the rectus muscle. It terminates by anastomosing with the superior deep epigastric artery.

2. *Superior Deep Epigastric Artery.*—The superior epigastric artery is a direct continuation of the internal mammary artery. It descends into the abdominal wall by entering the sheath of the rectus. It sends branches to the rectus muscle, and terminates by anastomosing with the inferior deep epigastric.

3. *Intercostal and Lumbar Arteries.*—These arise from the last intercostal, the subcostal (12th), and from the upper lumbar arteries. The main trunks arise at a much higher level than the terminal branches as found in the anterior abdominal wall. From their point of origin in the lumbar region they are prolonged forward between the internal oblique and the transverse abdominis muscles. Their terminal branches anastomose with each other and with the epigastric artery.

4. *Deep Circumflex Iliac Artery.*—The deep circumflex iliac artery springs from the lateral side of the external iliac. Its direction is toward the anterior superior spine, following the crest of the ilium. It ends by anastomosing with the iliolumbar artery.

5. *Cutaneous Vessels.*—The cutaneous vessels of the abdominal wall are the superficial circumflex iliac and epigastric. The iliac is a branch of the femoral. It is usually found along the line of the inguinal ligament, and ends in the skin surrounding the inguinal region. The superficial epigastric artery arises from the femoral also. Its direction is upward and medially toward the umbilicus. Neither of these vessels is of surgical importance. However, their relative position should be noted when making an incision, and immediately ligated to prevent undue loss of blood.

QUESTIONNAIRE

1. Why is a flexible abdominal wall essential?
2. Explain the relationship between intra-abdominal pressure and the abdominal wall.
3. What alters the contour of the abdomen?
4. Give the characteristics of a hypersthenic body habitus.
5. What are the characteristic features of the asthenic and hyposthenic types?
6. What is meant by "linea alba?"
7. What is the common name for it?
8. What is its surgical significance?
9. Where is the linea semilunaris located?
10. Describe its formation.
11. Why should it be avoided when making an abdominal incision?
12. What important vessel crosses the linea semilunaris below the umbilicus?
13. Name the "fixed landmarks" of the abdomen.
14. Define: (*a*) topographical point; (*b*) topographical line; and (*c*) topographical triangle. Give examples of each.
15. Give the various lines and planes of the abdomen.
16. Give the location and importance of the transpyloric plane.
17. Bound and give the surgical significance of the various diagnostic points of the abdomen.
18. Bound and give the surgical significance of Hesselbach's triangle.
19. Bound Petit's triangle; give its surgical significance.
20. Bound the superior lumbar triangle; give its surgical importance.
21. Bound the femoral triangle; give its surgical importance.
22. What fixed landmarks are recognized by Addison?
23. What is Addison's plane?
24. How would you determine Addison's point?
25. What structures are crossed by Addison's plane?
26. Describe Addison's method of abdominal topography.
27. How do other systems differ?
28. Name the various abdominal zones.
29. Give the origin, insertion, and relation of the following muscles: Rectus abdominis, internal oblique, external oblique, and transverse abdominis.
30. Give the origin and attachments of the lumbodorsal fascia.
31. From which layer do the latissimus dorsi and external oblique derive their fasciæ?
32. Give the origin and attachments of the transversalis fascia.
33. How is the rectus sheath formed?
34. How does it differ: (*a*) above the mid-hypogastric point; (*b*) below the mid-hypogastric point?
35. What is the semilunar fold of Douglas?
36. What is the relationship of this fold to the deep epigastric artery?
37. Give the origin and course of the deep epigastric artery.
38. What other arteries are found within the abdominal wall?

CHAPTER 42

THE PERITONEUM AND PERITONEAL CAVITY

By A. V. Partipilo

GENERAL CONSIDERATIONS

The peritoneum is a serous membrane lining the wall of the abdominal cavity and having extensive and intricate connections with every viscus of the abdomen. It is covered with delicate endothelial cells which normally secrete a fluid to prevent friction of the abdominal viscera. The peritoneum may also be regarded as a large lymph sac having great absorptive power. Liquids placed in the peritoneal cavity are generally absorbed into the blood stream, whereas solids are absorbed by the lymphatics.

Hertzler pointed out that the blood vessels are the true absorbents of the peritoneum. He found that when a dye is placed in the peritoneal cavity it will appear in the urine before it is seen in the lymphatics, thus proving that the dye goes through the general circulation before it makes its appearance in the lymphatics. Hertzler does not believe that any one area of the peritoneum has greater absorptive power than any other. Hence, he does not believe that Fowler's position has any physiological basis in the treatment of peritonitis. This is at variance with the generally accepted theory that absorption in the upper part of the abdomen is greater than that in the lower part.

Physiological Basis for Treatment of Peritonitis.—When the peritoneum is irritated it has the function of throwing out an abundant exudate, the cells of which are actively phagocytic. In disease the peritoneum assumes a defensive as well an an offensive role. The relative importance of these two functions is controversial and the choice of one or the other by the surgeon has a direct bearing upon his method of treating peritonitis. The "physiological surgeon" believes that the response of the peritoneum is mainly offensive, and for this reason he has adopted a conservative "watchful-waiting" attitude in the management of peritonitis. It is his belief that the phagocytes should be permitted to function with minimum of surgical disturbance. For instance, in acute peritonitis following appendicitis, the physiological surgeon will advise against operation, but will place the patient in bed in Fowler's position, giving nothing by mouth, retard intestinal movements, and wait until the acute active symptoms subside before operating. This is Ochsner's "starvation" method of treating peritonitis complicating appendicitis. This same school teaches that if the patient shows evidence of having a local abscess, the appendix is carefully removed, if easily accessible; if not easily accessible, the appendix is not removed, as this would entail breaking up adhesions which are looked upon as nature's wall of defense. There are some surgeons who, in the face of free pus, will remove the appendix and close the peritoneum tightly with drainage, believing that the peritoneum will be better able to cope with the condition without draining the "protective" exudate.

The second school of thought, of which H. W. Kennedy, of Philadelphia, was the most zealous exponent, takes the position that the reactions of the peritoneum to infection are defensive rather than offensive; that the peritoneum will absorb fluid, such as saline, but will reject irritating fluids by throwing out protective fluids and lymph in an effort to prevent absorption. This school teaches that it is not peritonitis, but its complications which determine the final outcome. With this thought in view, Kennedy believes that surgical interference necessitates evisceration of the involved structures, and that manipulation does not increase absorption or produce shock. He states, "we do not contend that infectious fluids placed within the abdominal cavity will not kill the patient. Such fluids will pass through the peritoneum and therefore might be classified as death due to peritonitis. This may be seen in frank perforations of the abdominal organs, such as perforations of the stomach or duodenum or possibly in a sudden perforation of the very large and distended appendix filled with black foul fluid, where there has been no effort by nature to prepare the peritoneum for such an overwhelming dose of infection." He further points out that these patients die before there is any evidence of peritonitis, hence being examples of deaths due to absence of peritonitis.

The surgical management of the peritonitic patient, from the standpoint of the above view, requires evisceration of the infected area and drainage in its broadest sense. Every source of infection in the peritoneal cavity is treated alike; be it omentum, appendix, the large or small bowel, diverticulum of Meckel, a pus tube, perforations of the stomach or duodenum, or perforations of the gall-bladder, the source of infection is reached and removed or surgically treated.

In order to prevent collapsing of the infected bowel, thus producing postoperative obstruction, drainage is brought about by the coffer-dam method. Drainage also means loosening of all adhered loops of bowel, since the cause of death in peritonitis is often due to intestinal obstruction. In conclusion Kennedy stated, "In fact, all the surgical steps in the treatment of peritonitic abdomen are based on the broadest principles of drainage, such as breaking of all adhesions, exposure of abscessed cavities, release of partial and complete obstruction of the bowel, plus the removal of all gangrenous structures including the removal of the primary source of infection which is the very foundation of drainage."

Volumes have been written on the subject of peritonitis and its surgical management. It would be extremely difficult and beyond the scope of this book to enter into a discussion of the relative merits of the various methods of treatment. The author believes that peritonitis, whether diffused or localized, demands immediate surgical intervention, and that the source of infection, in addition to the complications present, be properly and aggressively dealt with. The removal of the source of infection is not sufficient unless complications, such as crippling adhesions or obstruction of the bowel, are also treated.

While admitting the importance of the phagocytic function of the peritoneal exudate, it must also be stressed that in peritonitis the abdominal cavity is invaded with virulent organisms and toxic material which must be thoroughly and adequately drained. We do not hold the view that the peritoneum should be left undisturbed to fight its own battle. In the end it will lose this battle if some complication of peritonitis should develop, for in the final analysis it is the complication of peritonitis which determines the fatal issue.

The use of chemotherapeutic agents in the treatment of surgical infections have been increasingly effective during the past decade. In peritonitis all possible

advantage should be taken in the use of sulfonamides, penicillin and streptomycin, but the correction of systemic disturbances and the application of sound surgical principles in the treatment of complications must be kept in mind. Peritonitis produces a profound intoxication as a result of rapid absorption of bacterial toxins. Clinically the patient presents a picture of severe shock manifested by decreased blood volume, hypoprotienemia, destruction of red blood cells, and a disturbance in the fluid and electrolytic balance. The corrections of these disorders is of primary importance and they should not be abandoned in favor of penicillin or the sulfonamides as exclusive agents in the treatment of peritonitis. Finally, it is imperative to bear in mind that chemotherapeutic agents have not altered the virtues of well established fundamental surgical principles.

The General Cavity.—The terms "abdominal cavity" and "peritoneal cavity" must not be used synonymously. The former extends to the pelvic inlet, whereas the peritoneal cavity includes all the organs of the abdomen, in addition to those in the pelvic floor. The peritoneum is a membranous covering for the various organs, glands, blood and lymph vessels, and nerves. The covering may be complete for certain structures, but for others it merely stretches over them. Hence many structures, even though they belong to the general abdominal cavity, are nevertheless extraperitoneal. A good example is the pancreas. This organ is covered anteriorly with peritoneum, whereas its other sides are uncovered. Other extraperitoneal structures are: the third part of the duodenum, the kidneys, the pelvic colon, and the urinary bladder.

The peritoneum lining the abdominal wall on its anterior, lateral and posterior surfaces is known as the parietal peritoneum. In an abdominal operation it is through the anterior parietal peritoneum that the general cavity is entered.

The peritoneum covering the abdominal viscera is known as the visceral peritoneum or serosa. It is connected to the walls of the viscera by a layer of loose areolar connective tissue—tela subserosa. The amount of subperitoneal fat varies; in the upper part of the abdomen the amount is small, whereas in the pelvic region it is generally increased. This is necessary to allow greater mobility of the peritoneum for accommodating distention of the urinary bladder.

The visceral peritoneum, although intimately connected to the organs, is a part of and is continuous with the general abdominal peritoneum. Figure 239 illustrates the peritoneal relationship of various organs of the peritoneal cavity in the female. Note that many organs lying dorsal to the peritoneum are covered by this membrane on their ventral surfaces only. Other viscera, such as the small intestine, appear to have a complete investment of peritoneum and are suspended to the posterior body wall by a "mesentery," are known as intraperitoneal organs. The mesentery is composed of two layers of peritoneum containing lymphatic filaments, blood vessels, nerves, and a variable amount of fat. The whole circumference of the bowel is covered with peritoneum, except at its mesenteric attachment where a triangular space is formed. This space, called the mesenteric triangle, communicates with the retroperitoneal space. Thus, it is feasible for an infection in the mesenteric triangle to spread between the two layers of the mesentery to reach the retroperitoneal space.

Omenta.—When the peritoneum enfolds more than one organ so that it passes from one viscus to another without attachment to the body wall, it is spoken of as "omentum." The manner in which the stomach becomes attached to other viscera by the peritoneum (omenta) is of surgical importance. The stomach is completely

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covered with peritoneum (serosal layer) except along the lesser and greater curvature where the two layers emerge to form the lesser and greater omentum and the gastrosplenic ligament.

The lesser omentum is composed of two layers of peritoneum which originates from the inferior surface of the liver and passes to the lesser curvature of the stomach and duodenum. That portion of the omentum passing from the porta hepatis of the liver to the superior part of the duodenum is called the hepatoduodenal

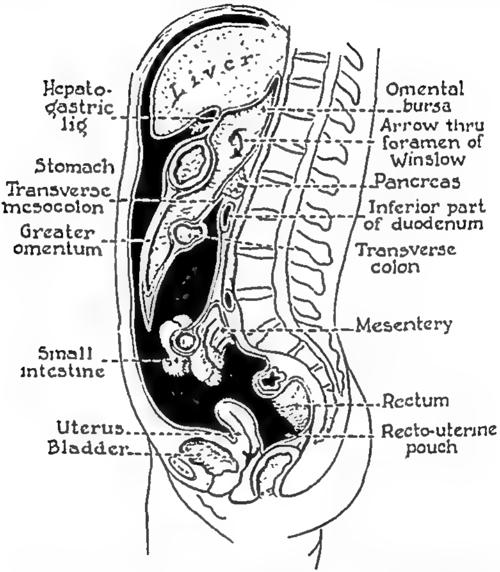


FIG. 239.—Diagrammatic median section of female body, to show the peritoneum on vertical tracing. The great peritoneal is black; the lesser omental bursa is darkly shaded; the peritoneum on section is shown as a white line; and a white arrow is passed through the foramen of Winslow from the great sac into the lesser sac (after Gray).

ligament. This ligament forms the anterior layer of the foramen of Winslow within which are located the common duct, hepatic artery, portal vein and lymphatic vessels. That portion of the lesser omentum between the liver and the stomach is called the *gastrohepatic ligament and contains the right and left gastric vessels,* lymph glands, and branches of the vagus nerves.

The two layers of the lesser omentum separate at the lesser curvature to form the anterior and posterior serosal covering of the stomach. They emerge at the

greater curvature and pass down in front of the transverse colon to become the anterior two layers of the greater omentum. That part of the omentum passing over the colon usually becomes attached to the transverse colon and is known as the gastrocolic ligament. They continue beyond the colon and are finally reflected back as the posterior two layers of the greater omentum. Thus the greater omentum is composed of four distinct peritoneal layers. The posterior two layers envelop the transverse colon and then divide so that one layer goes cephalad to form the posterior parietal peritoneum of the lesser omental bursa, while the other layer goes caudad to form the posterior parietal layer of the greater peritoneal cavity. The cephalad layer passes over the under surface of the diaphragm and then to the starting point at the inferior border of the liver.

The gastrosplenic ligament extending from the greater curvature of the stomach (fundus and body) to the spleen, consists of two layers of peritoneum which completely envelop the spleen. Within this ligament are found the vasa brevæ (short vessels) of the stomach derived from the splenic artery. The spleen is also supported by the liniorenal ligament and the phrenicocolic ligament. The latter is a triangular fold of peritoneum which attaches the splenic flexure of the colon to the diaphragm and permits the spleen to rest upon it.

The Lesser and Greater Omental Bursæ.—Thus, because of the manner in which it is formed, the lesser and greater omentum divides the peritoneal cavity into two sacs or bursæ: (1) Bursa omentalis, or the lesser sac of the peritoneum; and (2) the great sac, or the greater omental bursa. The great sac is opened when an incision is made in the abdominal wall. It encloses most of the abdominal viscera. The lesser sac lies behind the stomach and communicates with the larger sac through the foramen of Winslow. Thus anatomically the lesser omental bursa can be reached through the foramen of Winslow. A retrograde abscess, or the gastric contents of a ruptured ulcer on the posterior surface of the stomach, reaches the general cavity through this foramen. A retrograde abscess drains toward Morrison's pouch (subhepatic space) and it is for this reason that a drain is placed in this pouch.

Surgical approach to the lesser omental bursa may be obtained by any one of the following routes:

1. Through the lesser omentum. This route is seldom used, because it is difficult to reach any structure within the bursa.

2. Through the gastrocolic omentum. This is the favorite route to reach the pancreas, the second and third portion of the duodenum, and the posterior surface of the stomach. In cases of uncontrollable bleeding from gastric ulcers, the left gastric artery may be reached through this opening.

3. Through the transverse mesocolon. When an opening is made in the transverse mesocolon during a posterior gastrojejunostomy, the lesser sac is opened. A retrogastric abscess may also be drained through the mesocolon, however the preferable routes are through the foramen of Winslow and through the lesser omentum. The third portion of the duodenum can be exposed through the mesocolon, thus again opening the lesser sac.

4. Transgastric route. The lesser omental bursa is reached when a ruptured posterior ulcer is exposed through an anterior gastrotomy.

5. Through the posterior body wall. This route is seldom used, except to drain a bullet wound that has entered the lesser sac.

6. Through the chest wall. A left thoraco-abdominal incision enters the lesser

cavity. Through this incision the spleen and posterior surface of the stomach are easily reached.

Structures in the Subserous Layer of the Peritoneum.—1. Urachus.—This is the remains of the allantois. It is a slender band extending from the apex of the urinary bladder to the umbilicus. According to the researches of R. Campbell Begg, the urachus is rarely more than 5 centimeters in length. Its upper extremity is fully 11 to 12 centimeters inferior to the umbilicus. It is connected to the bladder *either by a single cord of fibrous tissue, or by diverging strands* which unite at the upper parts. These strands are known as Luska's plexus and are derived from the adventitious tissue of the umbilical arteries, teased out by the descent of the urachus.

2. Deep Epigastric Arteries.—The inferior deep epigastric artery arises from the external iliac artery, ¼ inch above the inguinal ligament at the femoral point. Its direction is upward and mesially toward the mid-hypogastric point where it enters the rectus sheath through the semilunar fold of Douglas. One is found on each side of the body.

The superior deep epigastric artery, a direct continuation of the internal mammary, descends into the abdominal wall and enters the rectus sheath. It anastomoses with the inferior epigastric.

3. Obliterated Hypogastric Artery.—Soon after birth the umbilical branch of the hypogastric artery atrophies and becomes converted into the lateral umbilical ligaments. *These spring from the hypogastric artery*, then run forward on each side of the pelvis to the apex of the bladder, finally ascending to the umbilicus, where they are attached.

4. Falciform Ligament of the Liver.—This is composed of two layers of peritoneum, within which are found the remains of the umbilical vein of the fetus It arises from the anterior abdominal wall and the diaphragm, descends in a crescentic (falciform) fold, attaching itself to the liver, and extends as far down as the umbilicus. *It is located immediately to the right of the mid-plane.* Its chief function is to mobilize and suspend the liver. In gall-bladder surgery the ligament is often utilized as a tractor to pull the liver out of the abdominal wound. An empyema of the gall-bladder or a liver abscess may spread to the abdominal wall through the ligament.

Peritonization.—In abdominal surgery one of the problems confronting the operator is the prevention of undue tears or abrasions of the peritoneum. Peritonization is the process of covering raw surfaces. The object is threefold: first, to assist primary healing of a wound created as a result of the operative maneuver; secondly, to prevent postoperative adhesions; and lastly, to prevent extention of an infection *into the retroperitoneal spaces.*

Peritonization as outlined by W. F. Lynch consists of four methods:

1. Raw surfaces are infolded beneath peritoneal flaps by suture; this method is used when the peritoneum is secured without undue traction. It is used in suturing intestine and stomach, in covering raw surfaces after pelvic operations and after appendectomy, and in suturing all free edges of the parietal peritoneum. This is the simplest and most useful method of peritonization. It should be employed *whenever possible.*

2. Cauterization.—In the strict sense of the word, this method is not a method of cauterization. Its object is to create a raw surface which is incapable of exuding fibrin; thus fibrous adhesions are prevented from adhering to structures This method does not have a wide application in abdominal surgery. It is applied

to denuded infected areas, to tags of redundant tissue, to cut surfaces of bowel, and over extensive oozing surfaces that cannot be covered with peritoneum. Needless to say, this method should be used with great caution.

3. Omental and Peritoneal Grafts.—The omental graft is used to cover extensive raw surfaces. It is usually secured from the greater omentum, laid over the area, and fixed in place by fine catgut sutures. The less fat content of the graft, the better the results. Peritoneal grafting is difficult because it is impossible to get a sufficient amount.

4. Peritonization by Covering With Neighboring Peritoneal Structures.—This method is of value when large areas are denuded and the use of omental or free peritoneal grafts is impractical. It is the method of choice in pelvic surgery when large areas are exposed.

QUESTIONNAIRE

1. What is the function of the peritoneum?
2. Discuss the physiology of absorption of peritoneal fluids.
3. What is Hertzler's theory of fluid absorption?
4. How does it differ with the generally accepted theory? How does it affect surgical thought?
5. Explain the role of the peritoneum when an infection takes place.
6. What is meant by "watchful waiting" treatment of peritonitis?
7. Explain Ochsner's "starvation" treatment of appendiceal peritonitis.
8. What is the view of the opposite school of thought in regard to the role of the peritoneum in peritonitis?
9. What is their view as to the final cause of death?
10. Give Kennedy's views as to the treatment of peritonitis. Explain.
11. Give a short résumé of the relative merits of the two schools of thought.
12. Discuss the value of chemotherapeutic agents in the treatment of peritonitis.
13. Define: peritoneal cavity and abdominal cavity.
14. What is meant by an extraperitoneal organ?
15. Define: (a) parietal peritoneum; (b) visceral peritoneum.
16. How is the "mesentery" formed?
17. Why is there an abundant subserous fat in the pelvic region?
18. How can an abscess in the mesenteric triangle reach the retroperitoneal space?
19. Define: tela subserosa.
20. Give the attachments of the stomach.
21. What structures are found within the hepaticoduodenal ligament?
22. How is the greater omentum formed?
23. Discuss the formation of the lesser and greater omental bursa.
24. [illegible]
25. [illegible] (a) urachus; [illegible] (d) falciform ligament of the liver.
26. Define peritonization.
27. Describe four methods of peritonization; explain the value of each.

CHAPTER 43

ABDOMINAL SECTION

By A. V. Partipilo

LAPAROTOMY

Definition.—Abdominal section signifies the act of cutting into the abdominal wall. Laparotomy may be defined as an incision into the loin. This comes from the Greek words lapara and tome, meaning an incision into the flank. By common usage the term signifies any exploratory incision made in the abdominal wall.

General Remarks.—The cardinal steps in every abdominal operation consists of making the incision, taking care of the lesion, and closing the abdominal wound. It can readily be seen that the making and the closing of the incision assume important roles in the success or failure of an operation. We must not lose sight of the fact that the process of opening and closing peritoneum is in itself a major surgical procedure. Very often it consumes the greater portion of the operating time. An ill-advised or improperly made incision will require more time and necessarily increase the operative risk.

Incisions vary according to the location of the suspected lesion and often there are numerous incisions for the same operation. Occasionally it is difficult to decide which is the best incision for a given operation. The surgeon should choose the simplest incision so that the maximum amount of freedom is gained for manipulating the underlying organs, without producing undue destruction of tissues. In acute appendicitis, for instance, a short McBurney incision made directly over the appendix is often satisfactory, but in chronic appendicitis, or when massive adhesions are present, such incision is inadequate, for it does not allow freedom of manipulation, nor does it allow sufficient exposure to other abdominal viscera. In such cases the appendix is removed, but the patient may continue to complain of the original symptoms.

A thorough anatomical knowledge of the abdominal wall is of primary importance for a properly executed incision. It is essential to have a fairly accurate knowledge of the location and the relationship of the various muscles, nerves, fasciæ, and blood vessels of the abdominal wall. In order to make the closure easier and more secure, the incision should be prepared for the closure as one enters the abdomen. In this manner an abdominal section is treated as though a hernia were present, and the closing is made according to the general principles of herniorrhaphy. A well-planned incision must take into consideration the following rules.

Rules in Abdominal Section.—1. Adequate Exposure: It is obvious that the incision should be made sufficiently long and wide to permit good exposure of the underlying organs. Incisions that require rough handling of the wound and prolonged forceful retraction are not justifiable. However, one should avoid a needlessly long incision, when a shorter one will suffice. G. A. Sloan, of Bloomington, found that the longer the incision is made, the greater the force required to bring the edges together. He estimated that the force required increases in propor-

tion to the square of the length of the incision. Nevertheless, the importance of gaining free access to the underlying structures outweighs any consideration of additional length in the incision. The incision should be made sufficiently long not only to provide easy access to the diseased part, but also to allow for complete exploration of the abdomen if indicated. An incision placed directly over the diseased part will give the most desired exposure, but a combination of lesions is a common finding, and in view of this the incision should be planned so that a co-existing pathological condition can be taken care of through the same wound.

2. Avoid Cutting Nerves: The nerve supply to the abdominal wall comes from the anterior rami of the lower six dorsal nerves. These enter the abdominal wall by piercing the transversalis and the internal oblique muscles, and pass forward to reach the sheath of the rectus, where each nerve divides into two branches. The larger branch proceeds beneath the muscle supplying its medial portion; the anterior branch innervating the lateral part of the muscle.

A well-planned incision safeguards the motor nerves to the rectus muscle. This is more important than cutting the muscle itself, for the muscle can be sutured without damage to its function. However, when possible, it is better to separate rather than cut its fibers across. When muscular fibers must be incised transversely, the cut ends should be carefully sutured at the completion of the operation. An upper abdominal median incision avoids the nerves; however, it goes through the thinnest portion of the abdominal wall and a weak scar is the usual result. On the other hand, an incision made at the lateral border of the rectus tends to destroy the nerves, thus predisposing to postoperative discomfort, loss of muscular tone, and development of a hernia.

Of the vertical incisions, a paramedian is advisable. It is made about ½ to 1 inch from the mid-line. After the anterior rectus sheath is incised, the muscle is retracted laterally, without disturbing its nerve supply. If any difficulty is encountered in freeing the muscle from the fascia, it may be split in the direction of its fibers with little damage. Incisions made through the middle of the rectus destroy part of the nerve supply, causing atrophy of the inner portion of the muscle.

Many surgeons advocate transverse incisions, asserting that they injure the nerves least. The objection has been made that any transverse division of the muscular or fascial fibers leaves a permanent weakness at the site of the division. Sloan avoids this by making the incision through the different layers in the direction of their fibers, the transversalis being incised transversely (see technique of Sloan's incision).

3. Avoid Cutting Blood Vessels: Whenever possible, the incision should be made so that the main blood supply to the rectus muscle is not interfered with. The chief blood vessels to avoid are the inferior and the superior epigastric arteries. They are found on the posterior and middle portions of the recti muscles. Whenever possible they should be protected. Depriving a muscle of its blood supply will undoubtedly retard its healing process, and ultimately its function. For this reason, avoid making an incision in the mid-rectus. A lateral rectus incision crossing the spino-umbilical line will encounter the inferior deep epigastric artery; it should be retracted out of the field, and not cut and ligated.

4. Hemostasis: Before opening the peritoneum make certain that all bleeding points have been ligated. Blood spilled into the peritoneal cavity may be the cause of postoperative adhesions, and it may also obscure the operating field. It is equally important to see that all intra-abdominal bleeding has been stopped before closing the abdominal wound.

Bevan Incision.—This incision begins just below the ensiform cartilage, runs downward and obliquely to about the middle of the rectus muscle, then vertically downward to about the level of the umbilicus, where it is carried obliquely outward and down for a distance of 2 or 3 inches. The fascia and muscle are split in the direction of their fibers. If necessary, the rectus muscle may be divided at the upper or at the lower part of the incision.

The Bevan incision is employed for surgery of the gall-bladder and stomach. In the majority of cases the oblique prolongations can be eliminated without greatly interfering with good exposure.

McArthur Incision.—The McArthur incision for gall-bladder surgery consists of a vertical splitting of the rectus muscle and a transverse incision of the transversalis and the peritoneum.

The usual paramedian incision is made through the skin, then the rectus fascia and muscle are retracted laterally. The transversalis is then incised transversely with the peritoneum. McArthur stresses the fact that the transversalis is an active respiratory muscle, and following longitudinal incision, with each respiration it so tugs and pulls on the line of sutures as to make it give way. He believes that by separating the fibers of the transversalis, instead of cutting them, this cannot occur. This incision does not provide adequate exposure to the stomach.

McBurney Incision.—The McBurney incision is made parallel with the fibers of the external oblique, about 1½ to 2 inches medial to the anterior superior spine of the ileum. It is about 2 to 3 inches long, with its center crossing the spino-umbilical line. The external oblique is incised in the direction of its fibers. The internal oblique and the transversalis muscles are likewise split in line with their course and retracted. The transversalis fascia and peritoneum are then divided in the direction of the fibers of the fascia.

This incision affords an opening about 2 inches in diameter, and it is placed immediately over the appendix. It permits direct access to the meso-appendix without the necessity of rotating the ileum or pulling on the cecum. A retrocecal appendix can be removed with greater ease through this incision than through a vertical one. On the other hand, difficulty may be encountered if the appendix is bound down with adhesions. It is inadvisable to carry out drainage through this incision; if necessary, drainage may be provided through a stab wound over the iliac crest, and the McBurney incision closed. The McBurney incision should not be used when the diagnosis is uncertain or exploration is contemplated.

To explore the pelvic organs in women, the skin incision may be extended downward and more mesially. After this, the aponeurosis of the external oblique is cut in the direction of its fibers to the same extent. The internal oblique and transversalis are then extended to the rectus muscle and the anterior sheath of the rectus is incised lengthwise and downward. The posterior sheath and peritoneum are likewise incised, after retracting the rectus muscle. In the lower angle of the incision the inferior deep epigastric artery may present itself; it must be retracted out of the field, if possible. The iliohypogastric nerve should be identified and every effort should be made not to include it in the suture when closing the incision.

Pfannenstiel Incision.—The Pfannenstiel is a combined transverse and longitudinal incision adapted for pelvic operations. The incision through the skin and fat is made transversely, with a slight convexity downward, the lowest point coming within 2 inches of the pubis. The anterior rectus sheath is also divided trans-

versely, then separated superiorly and inferiorly to free the recti muscles. The muscles are retracted laterally and a vertical incision is made through the thin posterior sheath, extraperitoneal fat and peritoneum.

The Pfannenstiel incision is well adapted for gynecological operations; however, if a large tumor is to be removed, or if the patient is obese and the abdomen pendulous, some difficulty may be encountered.

Sloan Incision.—The skin incision extends from the ensiform to a point 3 centimeters above the umbilicus, and continues outward on each side of the umbilicus. The flaps of skin and fat are dissected outward, exposing the aponeurosis of the inner borders of the recti muscles. Longitudinal incisions are made through the sheaths of the recti about 1 inch from the mid-line. The muscles, with the overlying fascia, fat, and skin, are retracted exposing the posterior sheath. The latter, with the peritoneum, is incised transversely from the outer edge of one rectus muscle to the outer edge of the other.

The Sloan incision is well adapted for surgery of all upper abdominal organs, including the spleen. The appendix can be exposed by downward and outward retraction.

TECHNIQUE IN MAKING AN ABDOMINAL INCISION

For the purpose of showing the technique in making an abdominal incision, the right paramedian incision will be described.

The patient is placed in the correct position on the operating table, the skin is then prepared in the usual fashion, and the operating field is adequately draped.

The proposed line of incision is made through the skin and subcutaneous tissue from the tip of the ensiform, obliquely downward about one inch to the right of the linea alba and carried vertically downward to the level of the umbilicus. Tiny scratches may be made with a needle at a right angle to the proposed line of incision to indicate the points of apposition when closing; this will result in a finer and firmer linear scar.

The surgeon should decide beforehand the length of the incision. Long incisions, as a rule, are better than short ones, otherwise considerable retraction will be required to obtain proper exposure. Longer incisions heal just as well as short ones, and they eliminate the likelihood of tearing and bruising the tissues. It is difficult to say how long an incision should be. For operations requiring a great deal of manipulation, the incision should be long enough to admit the entire hand through the wound. Figure 241, *c*, demonstrates the author's method of determining the length of the proposed line of incision. The palm of the hand is placed on the abdomen and the incision is measured to extend 1 inch on either side of the hand. In this manner it is reasonably certain that the length will be sufficient to admit the operator's hand into the abdominal cavity. The surgeon must also take into consideration the obesity of the patient; if the patient is obese, the incision is made slightly longer.

With one hand steadying the skin, as illustrated in Figure 241, *a*, the incision is made with one stroke of the scalpel. Notice that countertraction on the skin is made opposite the cutting surface, rather than by lateral traction. This method will insure a straight incision; this is especially important in plastic surgery, or in surgery of the exposed surfaces. Avoid bevelling of the skin edges by holding the scalpel at a right angle to the cutting surface (Fig. 241, *b*). Both corners of the skin and subcutaneous tissue should be plumb with the skin cut (Fig. 242, *a*). Too

often the surgeon deprives himself of considerable space by making the incision gutter-shaped.

Before proceeding with the incision all bleeding points should be clamped and ligated, with fine plain catgut, silk or cotton. Oozing surfaces may be controlled with hot saline packs. For small bleeders forci-pressure, or forci-pressure combined with torsion, may suffice. After all bleeding points have been controlled, towels may be fixed to the skin edges by special towel clips. The scalpel which was used for the skin incision should be discarded, because it has cut through unsterilized deeper layers of the skin.

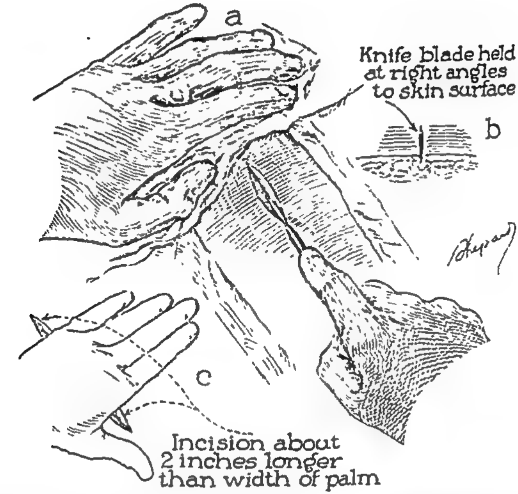


FIG 241.—The incision through the skin; *a*, the operator uses the left hand for counterpressure; *b*, cutting is done at right angle to the wound; *c*, method of measuring the length of the proposed line of incision.

The fascia is now cleared of fat along the proposed line of incision, and with a new scalpel a small opening is made and the fascial incision is completed with scissors as illustrated in Figure 242, *b*. With sharp dissection the edges of the fascia are freed from the muscular attachments for the entire length of the wound. This is an important part of the procedure; an anatomical dissection is made in order to prepare the fascia for its subsequent closure at the termination of the operation (Fig. 242, *d*).

With sharp dissection the rectus muscle is separated vertically throughout the entire length of the wound (Fig. 242, *e*). *Before proceeding* with the incision, ligate all bleeding points.

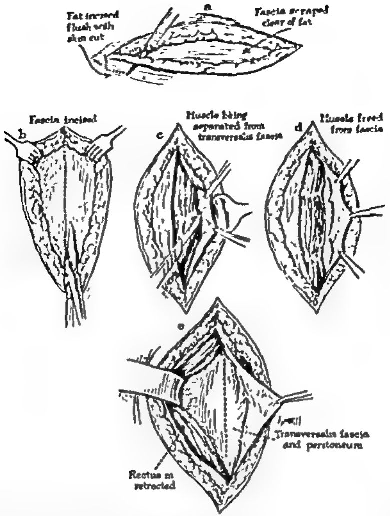


FIG. 242.—*a*, The subcutaneous tissue is incised plumb with the skin cut; *b*, *c*, *d*, illustrate the method of freeing the rectus fascia from the underlying muscle; *e*, lateral retraction of the rectus muscle, exposing the transversalis fascia and peritoneum.

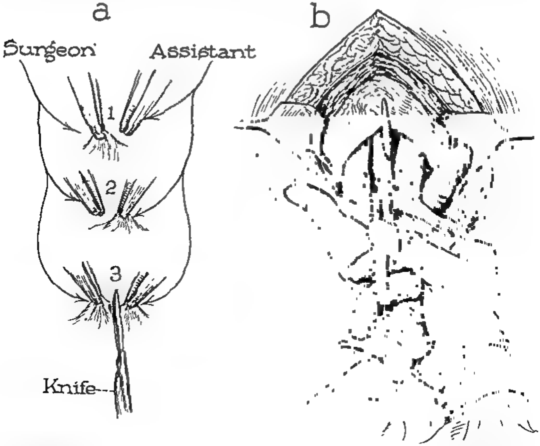


FIG. 243.—*a*, Author's method of picking up the peritoneum to avoid including any underlying structure; *b*, the peritoneum is incised with scissors; the fingers guiding against injury to intra-abdominal structures.

With the edges of the wound held apart, the peritoneum is picked up with tissue forceps; one is held by the assistant, another in the left hand of the operator. Before cutting, the surgeon should guard against injury to any structure that may be accidentally picked up with the forceps. Figure 243 illustrates the author's method of avoiding injury to the underlying structures. The peritoneum is first grasped and held up; then the assistant takes hold at a point less than ½ inch away. The surgeon now releases his end and regrasps the peritoneum; the assistant repeats

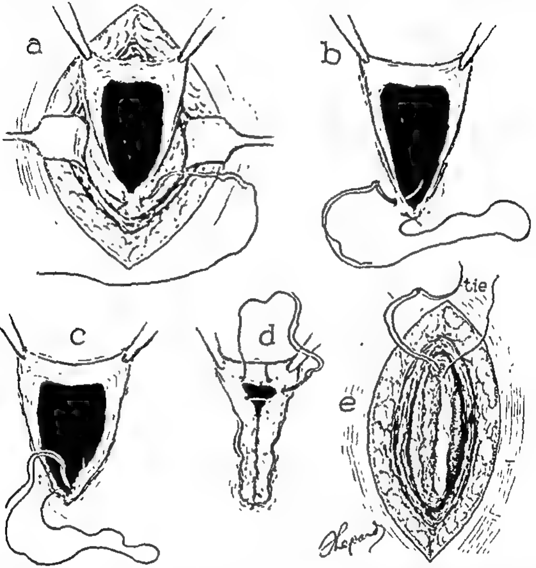


FIG. 244.—Closure of the peritoneum; *a*, anchor stitch; *b*, the needle re-enters the peritoneal cavity; *c*, shows application of the Cushing type of suture; *d*, method of ending suture; *e*, the suture is tied

his maneuver. In this manner any underlying structure which may be caught within the forceps of the surgeon or the assistant at the first application of the forceps will become free and not apt to be picked up by the second grasp.

With a clean knife the peritoneum is opened for a distance of 1 inch and the incision is completed with scissors. As illustrated in Figure 243, *b*, the fingers act as guides when cutting with scissors. If any structure such as the omentum is adherent to the peritoneum, the incision is deferred until this is freed.

In the region of the mid-rectus the posterior rectus sheath is closely fused with the peritoneum, except in very obese individuals. In cases where the amount

of fat is minimal or absent the sheath and the peritoneum should be opened as one layer. Even in thin patients, there is considerable amount of preperitoneal fat in the upper and lower part of the median line. In such cases the posterior rectus sheath is completely separated from the peritoneum.

After the abdominal incision is completed, a preliminary exploration is made and a self-retaining retractor is applied to hold the wound apart.

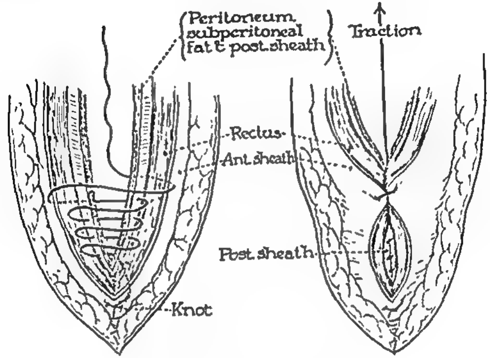


Fig. 215.—Pool's "Zipper Stitch."

CLOSURE OF THE ABDOMINAL INCISION

The incision just described is closed in the opposite manner from which it was begun. Before removing the retractor, the surgeon should make a final inspection for bleeding, and check and verify the sponge count.

Both angles of the peritoneum are grasped with Allis forceps and two more forceps are applied on its edges. Avoid using ordinary hemostats as they crush the peritoneum unnecessarily, thereby favoring the formation of adhesions. The assistant holds the peritoneum upwards and at the same time everts the edges. With the peritoneum thus held, the operator sutures it with plain catgut No. 0 threaded on a non-cutting sharply curved needle. Eversion of the peritoneum may be produced by using a Cushing type of suture, as illustrated in Figure 244. Two or three bites may be taken and left loose; then it is pulled taut in the direction of the incision. If the peritoneum is torn, ragged, or difficult to close, the suture can include the posterior rectus sheath and, if necessary, the fibers of the posterior part of the rectus muscle may also be included.

In some cases the "Zipper Stitch" advocated by E. H. Pool, of New York, may be used with advantage. His method is as follows:

With a double strand of chromic catgut No. 1, the anterior rectus sheath is picked up below the lower angle of the wound and tied. The needle is then passed through the rectus and posterior sheath, including the peritoneum. It is then passed back and forth from one edge of the posterior sheath to the other three or

four times and *left* very loose. *Next*, the needle is passed forward through the rectus and anterior sheath across the wound and inward through the opposite anterior sheath, rectus, posterior sheath, and peritoneum. Then the suture is slowly pulled taut in the direction of the incision, and not at an angle (Fig. 245). *The strain is taken up by the fascia and the rectus without straining the peritoneum.* The suture is continued until the entire peritoneal surface is coapted.

Printy recommended interrupted mattress sutures as a method of closing the peritoneum (Fig. 246). After the mattress sutures are applied, a continuous suture is inserted in an over-and-over fashion. The mattress sutures facilitate suturing, relieve tension, and reinforce the closure.

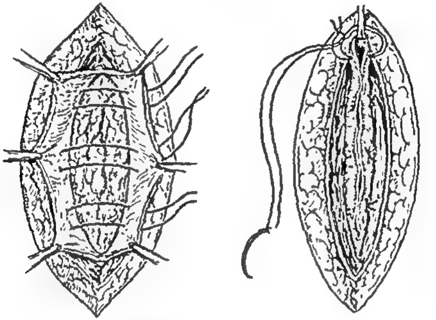


FIG. 246.—Printy's method for closing the peritoneum; the application of mattress sutures and the beginning of a continuous suture.

The posterior rectus sheath is next sutured with a continuous or preferably interrupted chromic catgut No. 0. Silk or cotton may be employed, however these must be used as interrupted sutures. Avoid puncturing muscular vessels. Such injury is often overlooked, with the result that a large hematoma develops beneath the muscle. In many cases where the posterior sheath is thinned out, it may be sutured together with the peritoneum.

The next procedure is to sew the anterior rectus sheath. Allis forceps are applied at the two angles and mid-way on each edge of the fascia. The suture is a continuous or interrupted chromic catgut No. 0 threaded on a curved needle. Interrupted cotton or silk sutures may be used instead of catgut.

Figure 247 illustrates four different methods of applying tension sutures. These are inserted through the skin and subcutaneous fat, and may also include the edges of the sutured fascia. Figure 247, *d* illustrates a continuous tension and approximating suture which is especially adapted for obese patients.

The skin may be sutured with a variety of methods as illustrated in the accompanying diagrams. The author prefers the simple continuous dermal suture (Fig. 248). The stitches are applied about three-eighths of an inch apart. They are pulled tight enough to bring the edges together without protrusion of fat or inversion

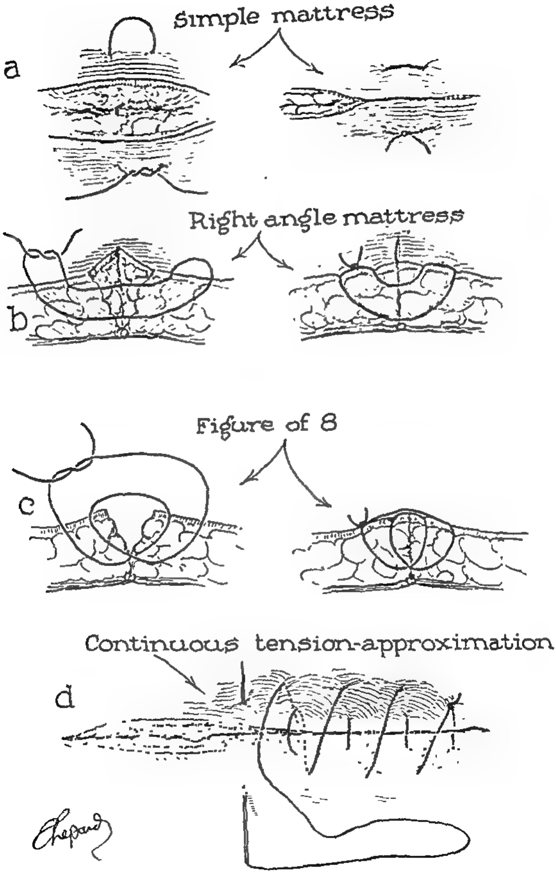


FIG. 247.—Various types of tension sutures.

of the skin edges. Figure 249 illustrates the author's method of inserting the needle to prevent inversion of the skin; the entire thickness of the skin, together with the fat is grasped with a tissue forceps and the needle is thrust through (Fig. 249, *c*); the other side is then similarly grasped and the needle is thrust through at a point corresponding to the site of emergence of the first bite (Fig. 249, *d*). As illustrated

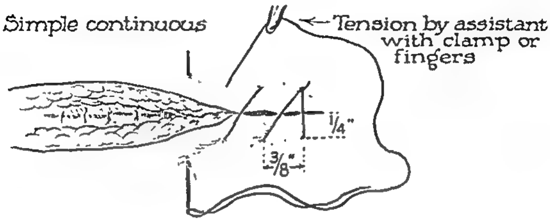


Fig. 248.—Simple continuous suture.

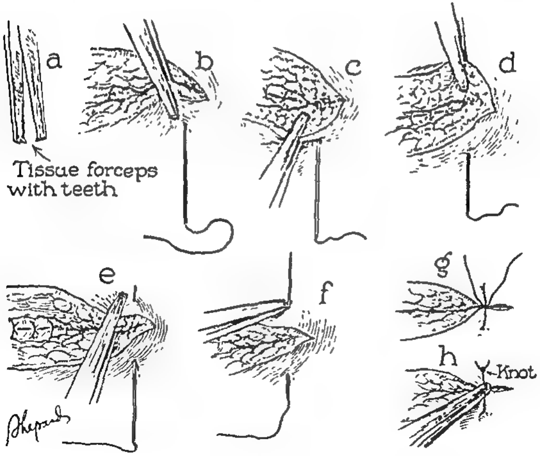


Fig. 249.—Method of inserting the needle for suturing the skin

in Figure 249, *f*, the needle may be pulled through with greater ease by applying counterpressure with tissue forceps. When the suture is pulled taut, the skin edges will be brought together in perfect apposition. Inversion is prevented by the layer of fat which overlies the suture. Figure 249, *g* illustrates the wrong position of the knot, and Figure 249, *h* the right position.

Besides the simple continuous suture for closing the skin, other methods have been described, such as continuous-lock, interrupted, shoe-lace, metal clips, etc. Figure 250 illustrates a method of applying an intermediate knot to a continuous suture. This knot is especially valuable when placed on either side of a drainage tube. Figure 251 illustrates two other methods of tying after completion of a suture.

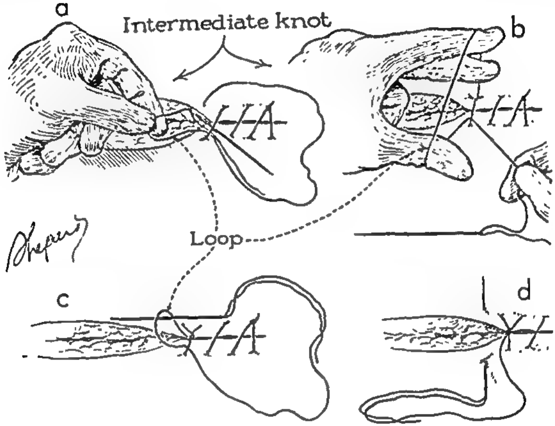


FIG. 250.—A method of interrupting a continuous suture by knotting at intervals

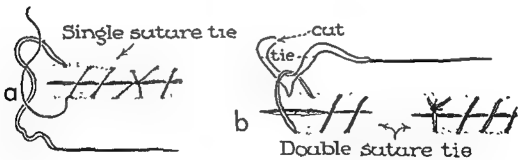


FIG. 251.—Methods for completing and tying with: *a*, single suture; *b*, double suture.

Figure 252 illustrates all the essential steps for the application of the subcuticular stitch. Figure 253 shows Reder's end-tie for the beginning and the end of a subcuticular stitch. The author's method is shown in Figures 252, *f* and *g*.

After the skin is closed, the wound is cleansed with alcohol and a light gauze roll is laid over it. At this time the traction stitches are tied over the gauze, thus completing the closure of the abdominal incision. Dressings are applied and the patient is returned to the ward.

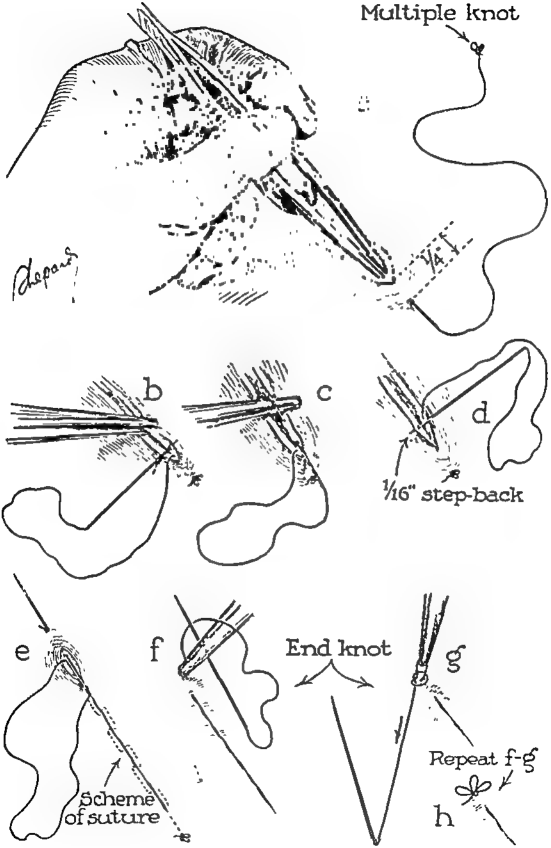


Fig. 252.—Subcuticular stitch and method of securing ends.

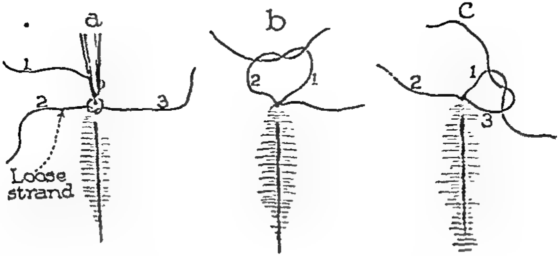


FIG. 253.—Reder's end-tie for subcuticular stitch.

SLOAN'S TRANSVERSE INCISION

An incision is made through the skin and subcutaneous tissue down to the fascia extending from the ensiform to a point about 3.5 centimeters above the umbilicus. It is continued outward and downward on either side of the umbilicus; it may be extended to about 4 centimeters below the umbilicus, if desired (Fig. 254). Thus an inverted V-shaped piece of skin and subcutaneous tissue is left around the umbilicus. A flap of skin and fat is dissected outward, exposing the

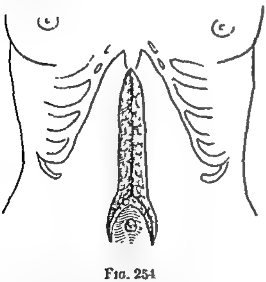


FIG. 254

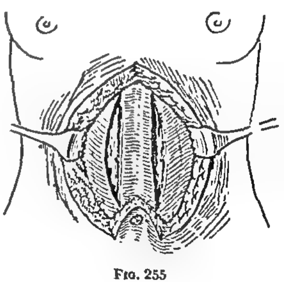


FIG. 255

FIG. 254.—Sloan's skin incision.

FIG. 255.—Vertical incisions through the external sheaths made 1 centimeter external to the inner border of the recti muscles.

aponeurosis over the inner borders of both recti muscles. Vertical incisions are then made through the fasciæ about 1 centimeter lateral (Fig. 255). The longer these incisions are made, the greater the amount of exposure which will be obtained.

The recti muscles, with the overlying skin and fat, are rolled outward and held by suitable retractors (Fig. 256). A transverse incision is now made through the exposed posterior rectus sheath and the peritoneum. It is extended across the linea alba parallel to the direction of the fibers from the outer edge of one rectus to the outer edge of the others. Lateral and vertical retraction affords an opening

with a diameter approximately equal to the length of the incisions in the anterior sheaths. This gives ample approach for all operations in the upper abdomen. The opening can be retracted over the regions of the gall-bladder, spleen, or the appendix.

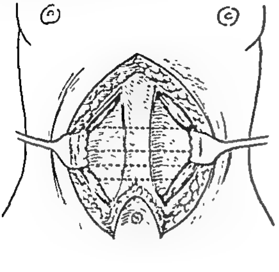


FIG. 256

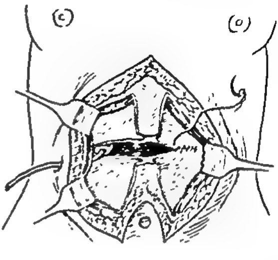


FIG. 257

FIG. 256.—The rectus muscle with the overlying sheath, fat and skin are rolled outward and held by suitable retractors, exposing the posterior sheath of the rectus. Dotted lines indicate the sites that may be chosen for the transverse incision through the aponeurosis and peritoneum.

FIG. 257.—In cholecystostomy the drainage tube is brought out through a puncture wound at the outer border of the rectus.

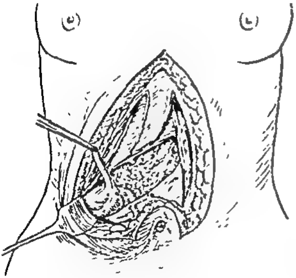


FIG. 258

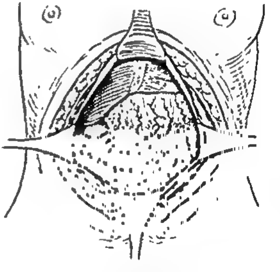


FIG. 259

FIG. 258.—The appendix is more readily dealt with through this incision than through a high

For Operations on the Gall-bladder.—Retraction upward and outward affords access to the region of the gall-bladder, liver, and hepatic flexure of the colon. For cholecystectomy and cholecystostomy, the right half of this incision is all that is usually required. An evident advantage is the ease with which one-half of this incision can be converted into the larger one. When drainage is required, the tube can be brought out through a puncture wound as illustrated in Figure 257.

For Appendectomy.—Retraction downward and outward affords access to the region of the appendix (Fig. 258). The appendix is more easily dealt with through this incision than through a high right rectus incision.

CLOSURE OF SLOAN'S INCISION

The peritoneum is closed with plain catgut No. 0, as in the vertical rectus incision previously described. It is begun at the outer end of the transverse incision and completed as illustrated in Figure 260-*A*. The posterior sheath is closed with interrupted silk or cotton sutures.

After the transverse incision is closed, the tension is taken off all other structures, hence the incisions of the anterior sheaths can be closed without any difficulty (Fig. 260-*B*). Sloan stresses the fact that the rectus muscle is firmly attached to the anterior sheath and not to the posterior. Therefore, during coughing, vomiting, or straining, contraction of the rectus pulls the flap of the anterior sheath inward and relieves the suture line of any strain.

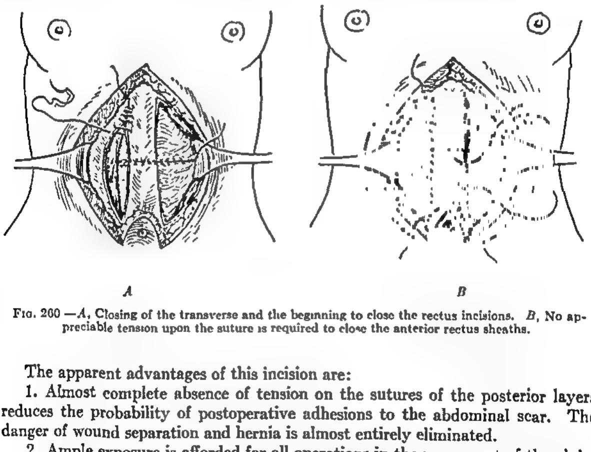


FIG. 260.—*A*, Closing of the transverse and the beginning to close the rectus incisions. *B*, No appreciable tension upon the suture is required to close the anterior rectus sheaths.

The apparent advantages of this incision are:

1. Almost complete absence of tension on the sutures of the posterior layers reduces the probability of postoperative adhesions to the abdominal scar. The danger of wound separation and hernia is almost entirely eliminated.

2. Ample exposure is afforded for all operations in the upper part of the abdomen. The author has found this incision completely satisfactory for partial gastrectomy; it permits easier mobilization of the duodenum, which is rather difficult with the usual vertical incisions.

3. Injury to the nerves and blood vessels is avoided.

4. Closure is accomplished with greater ease. It is surprising how easily the peritoneum is closed. The anterior fascial layers also are closed without strain.

QUESTIONNAIRE

1. Define: (*a*) abdominal section; (*b*) laparotomy.
2. What is the common meaning of the word "laparotomy"?

3. What are the three cardinal steps of any abdominal operation?
4. What are the objections to a long incision?
5. Give the rules in abdominal section.
6. Discuss the importance of each.
7. What factors must one bear in mind when closing an incision?
8. Define: median incision.
9. Give its advantages.
10. Give its disadvantages.
11. Define: paramedian incision.
12. When is a paramedian incision employed?
13. What are the objectionable features of a lateral rectus incision?
14. Describe Bevan's incision.
15. When is it employed?
16. What is McArthur's incision?
17. When is it employed?
18. Describe McBurney's incision.
19. When is it indicated?
20. What are the contraindications to a McBurney incision?
21. To explore the pelvic organs, how can the incision be extended?
22. What vessel may be encountered when making McBurney incision?
23. What is a Pfannenstiel incision?
24. When is it employed?
25. Describe the technique of Sloan's transverse incision.
26. For ordinary purposes, how would you determine the length of an incision?
27. How would you avoid bevelling the skin?
28. Why should the scalpel be discarded after making the skin incision?
29. Why should fascial flaps be dissected free?
30. How would you control a muscular bleeder?
31. How would you control bleeding points or oozing in the subcutaneous tissue?
32. What precautions are taken when opening the peritoneum?
33. Before closing the peritoneum, what two important precautions must be taken?
34. Describe the various methods of closing the peritoneum.
35. What is Pool's "Zipper Stitch"?
36. What is the advantage of using interrupted mattress suture for closing the peritoneum?
37. How is the posterior rectus sheath treated?
38. What is a tension suture?
39. When is it indicated?
40. Describe the following: (*a*) simple mattress; (*b*) right angle mattress; (*c*) Figure-of-8 mattress; and (*d*) continuous tension-approximating suture.
41. Describe the technique for suturing the skin without producing inversion of the skin edges.
42. Give the various methods of closing the skin.
43. Describe Reder's end-tie.
44. What are the advantages of Sloan's incision?

CHAPTER 44

INGUINAL HERNIA

By A. V. Partipilo

ANATOMY OF THE INGUINAL REGION

Descent of the Testis.—The cause of the descent of the testis is still a subject of dispute. The following description closely follows Cunningham: In early fetal life the testis is located on the posterior wall of the abdomen at the level of the upper lumbar vertebræ, and just below the level assumed at this time by the permanent

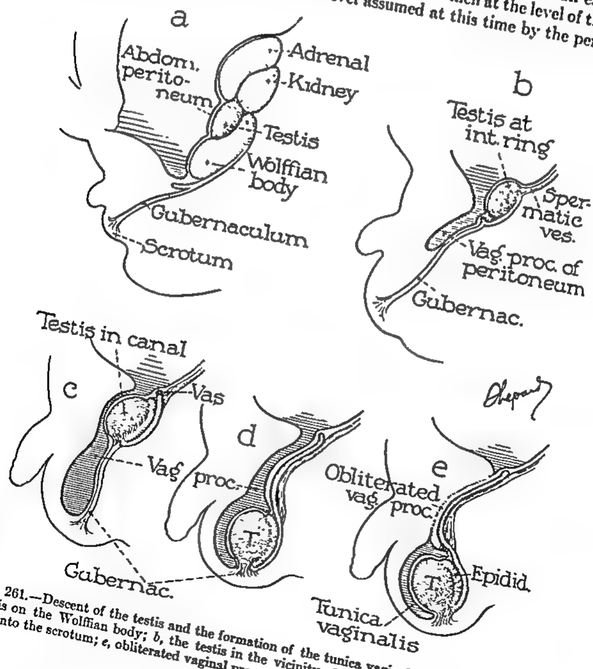


Fig. 261.—Descent of the testis and the formation of the tunica vaginalis. *a*, The origin of the testis on the Wolffian body; *b*, the testis in the vicinity of the abdominal ring; *c*, *d*, final descent into the scrotum; *e*, obliterated vaginal process, called the ligamentum vaginalis.

3. What are the three cardinal steps of any abdominal operation?
4. What are the objections to a long incision?
5. Give the rules in abdominal section.
6. Discuss the importance of each.
7. What factors must one bear in mind when closing an incision?
8. Define: median incision.
9. Give its advantages.
10. Give its disadvantages.
11. Define: paramedian incision.
12. When is a paramedian incision employed?
13. What are the objectionable features of a lateral rectus incision?
14. Describe Bevan's incision.
15. When is it employed?
16. What is McArthur's incision?
17. When is it employed?
18. Describe McBurney's incision.
19. When is it indicated?
20. What are the contraindications to a McBurney incision?
21. To explore the pelvic organs, how can the incision be extended?
22. What vessel may be encountered when making McBurney incision?
23. What is a Pfannenstiel incision?
24. When is it employed?
25. Describe the technique of Sloan's transverse incision.
26. For ordinary purposes, how would you determine the length of an incision?
27. How would you avoid bevelling the skin?
28. Why should the scalpel be discarded after making the skin incision?
29. Why should fascial flaps be dissected free?
30. How would you control a muscular bleeder?
31. How would you control bleeding points or oozing in the subcutaneous tissue?
32. What precautions are taken when opening the peritoneum?
33. Before closing the peritoneum, what two important precautions must be taken?
34. Describe the various methods of closing the peritoneum.
35. What is Pool's "Zipper Stitch"?
36. What is the advantage of using interrupted mattress suture for closing the peritoneum?
37. How is the posterior rectus sheath treated?
38. What is a tension suture?
39. When is it indicated?
40. Describe the following: (*a*) simple mattress; (*b*) right angle mattress; (*c*) Figure-of-8 mattress; and (*d*) continuous tension-approximating suture.
41. Describe the technique for suturing the skin without producing inversion of the skin edges.
42. Give the various methods of closing the skin.
43. Describe Reder's end-tie.
44. What are the advantages of Sloan's incision?

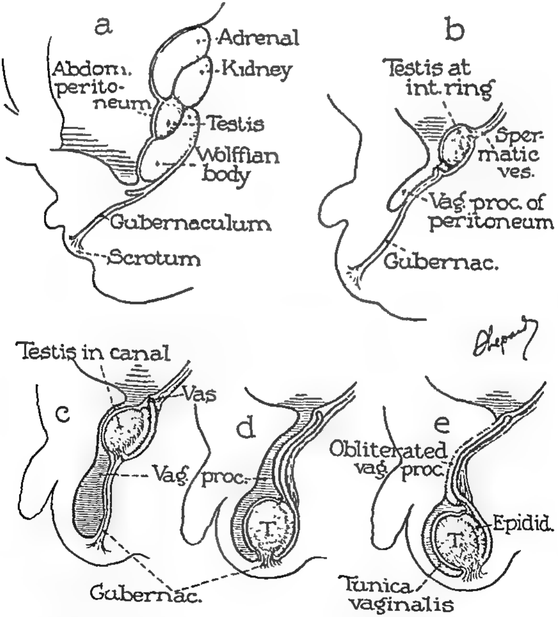
CHAPTER 44

INGUINAL HERNIA

By A. V. Partipilo

ANATOMY OF THE INGUINAL REGION

Descent of the Testis.—The cause of the descent of the testis is still a subject of dispute. The following description closely follows Cunningham: *In early fetal life the testis is located on the posterior wall of the abdomen at the level of the upper lumbar vertebræ, and just below the level assumed at this time by the permanent*



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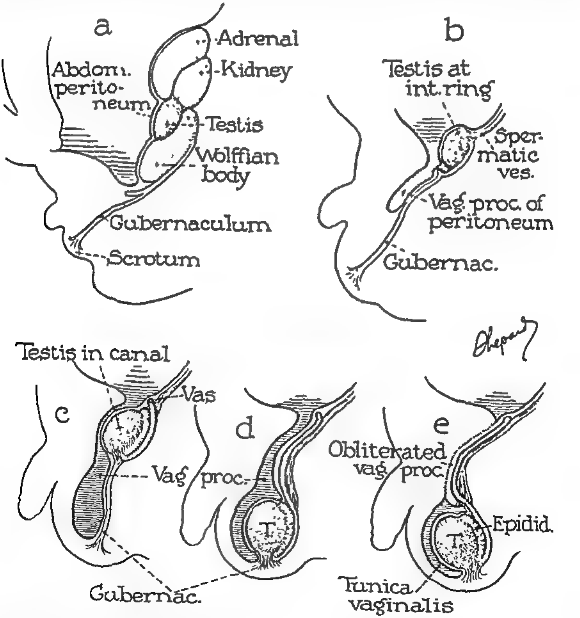

Fig. 261.—Descent of the testis and the formation of the tunica vaginalis. *a*, The origin of the testis on the Wolffian body; *b*, the testis in the vicinity of the abdominal ring; *c*, *d*, final descent into the scrotum; *e*, obliterated vaginal process, called the ligamentum vaginalis.

kidney, and is held in place by a fold of peritoneum or mesentery, called the mesorchium. As growth goes on, the testis is found to occupy a lower level in the abdominal cavity; in the third month it lies in the iliac fossa, and in the seventh it is situated near the abdominal inguinal ring (Fig. 261, *b*). Meanwhile a blind pouch of the peritoneum, termed the vaginal process, has grown downwards and medially through the anterior abdominal wall towards the scrotum, deriving as it goes a covering from each of the layers of the abdominal wall through which it passes. The testis with its mesorchium enters the vaginal process and descends within it until the scrotum is reached. At a later stage, the connection between the part of the vaginal process that lies in the scrotum and the peritoneal lining of the abdomen, becomes lost by the obliteration of the upper part of the pouch (Fig. 261, *b*). That part of the vaginal process that persists in the scrotum becomes the parietal layer of the tunica vaginalis, while the visceral layer is the primitive peritoneal covering of the testis and epididymis.

In the adult, a small fibrous band—the "ligamentum vaginale"—may be found passing through the inguinal canal and joining the peritoneum superiorly in the region of the internal abdominal ring. Sometimes this band is connected below with the tunica vaginalis, but more often it cannot be traced so far downwards. If the vaginal process does not become obliterated, a variety of "congenital" types of hydrocele, hernia, or combination of both, may result.

It is considered by some anatomists that the downward movement of the testis is due partly to a pull caused by the shrinking of the gubernaculum as it atrophies. The gubernaculum, when fully developed (about the sixth month), is attached above to the lower end of the testis, while inferiorly it is fixed near the inguinal region. In the lower part of its course it is related closely to, and is covered partly by, the peritoneum of the vaginal process. As the testis enters the vaginal process the gubernaculum atrophies, but at birth a short part of the gubernaculum may still be found passing downwards towards the inferior part of the scrotum and lying below the level of the tunica vaginalis.

The Inguinal Canal.—The inguinal canal is an oblique space transmitting the spermatic cord in the male or the round ligament in the female. It measures about 4 centimeters in length and extends from the internal to the external abdominal rings. The canal is longer in the female because the pelvis is considerably broader and the symphysis is higher. The obliquity of the canal (Fig. 262) constitutes a natural obstacle to the formation of hernia. This is due to the fact that any increase in intra-abdominal pressure forces the walls of the canal closer together.

The Internal Abdominal Ring.—The internal abdominal ring is situated at about the mid-point and $\frac{1}{2}$ inch medial to the inguinal ligament. It has been noted that when the testis descends, it pushes ahead the transversalis fascia; hence the abdominal inguinal ring (internal ring) is an aperture in the transversalis fascia. Its fibers are prolonged over the spermatic cord and the testis, enclosing them in a distinct pouch. This is known as the infundibuliform or the internal spermatic fascia. It constitutes one of the coverings of an oblique inguinal hernia. Besides the transversalis, the spermatic cord is invested with fascial layers from the internal oblique (cremasteric fascia) and the external oblique (external spermatic fascia). The deep inferior epigastric artery ascends obliquely along the inner margin of the internal abdominal ring, where it lies between the transversalis fascia and the peritoneum.

External Abdominal Ring.—The external abdominal ring (also called the superficial or subcutaneous abdominal ring) is an aperture in the aponeurosis of the external oblique for the passage of the spermatic cord. A thin layer of the fascia is prolonged downward upon the spermatic cord. This is called the external (intercolumnar) spermatic fascia.

The aperture is not strictly a "ring," but is triangular in shape, and oblique in its direction (Fig. 263). The pubic spine and crest form the base and the decussated intercolumnar fibers form the two sides. On the margins are found the

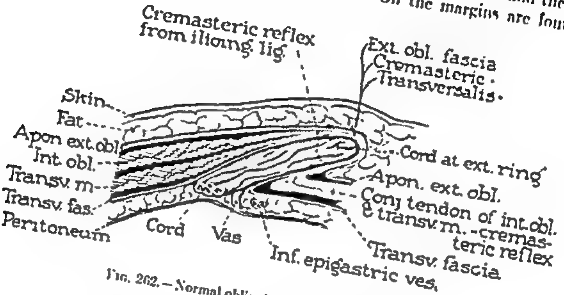

Fig. 262.—Normal obliquity of the inguinal canal.

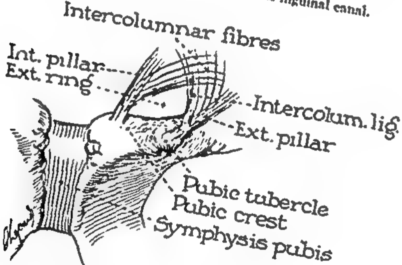

Fig. 263.—External abdominal ring. Illustrating the triangular and the interlacing fibers.

pillars of the ring. The external pillar, inserted into the pubic tubercle, is narrow, and forms a groove upon which the spermatic cord rests. The internal pillar lies medial to the cord and is attached to the crest of the pubis. According to Iason, the interlacing position of the fibers is for the purpose of diminishing the size of the ring by approximating the crura, thereby shortening the antero-posterior diameter of the ring. This can be demonstrated by inserting the examining finger into the external abdominal ring. When the patient coughs, the constricting action of the fibers at the ring is readily elicited.

The aperture measures about 1 inch from the base to the apex, and ½ inch from side to side. Because the normal ring will seldom admit the tip of the examining finger, it is usually impossible to palpate a normal inguinal ring.

Contents of the Inguinal Canal.—The inguinal canal contains the spermatic cord, the external spermatic vessels, external spermatic nerve, ilio-inguinal nerve, ilio-hypogastric nerve, and the obliterated vaginal process.

Spermatic Cord.—Normally the spermatic cord takes up the space of the inguinal canal. It is composed of the following structures:

1. *The Ductus Deferens.*—This is the most important structure of the cord. It is distinguished by its hard, cord-like consistency when the cord is held between the index finger and the thumb. It lies posterior to the cord, behind the veins and arteries. It is often intimately adherent to the sac and its separation may be difficult. Care must be taken to avoid cutting it. If accidentally divided, the two ends can be sutured with a fine Cambric needle threaded with black silk, using the same technique as in suturing a divided vessel. Watson recommends passing catgut thread through the lumen of each end, and tying a knot in each end of the thread (Fig. 264).

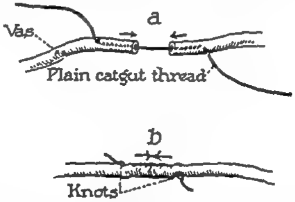

FIG. 264.—Method advocated by Watson for repairing a divided vas deferens

2. *Arteries.*—The external and internal spermatic arteries and the artery to the ductus deferens are found within the cord. The external spermatic artery is a branch of the inferior epigastric. It is a small twig supplying the cremasteric and anastomoses with the internal spermatic artery. The latter arises within the abdomen from the front of the aorta, entering the inguinal canal and then proceeding to the testis. The spermatic artery can be located by feeling its pulsations in the cord. It should never be divided unnecessarily. Cases are on record where atrophy and necrosis of the testis have resulted from severing the artery.

3. *Pampiniform Plexus of Veins.*—These arise from the posterior border of the testis, and as they pass upward form in the cord a bulky plexus. From this a single vessel issues into the abdomen through the internal abdominal ring. On the right side it enters the inferior vena cava; on the left it joins the left renal vein. A varicose condition of these veins gives rise to a condition known as varicocele.

4. *Lymph vessels.*—The spermatic lymph vessels enter the abdomen through the internal ring and join the lumbar glands.

5. *External Spermatic Nerve.*—This is a branch of the genito-femoral supplying the cremasteric muscle. When stimulated, reflexively the testis is pulled upward by the action of the cremasteric muscle. There are also sympathetic twigs which extend downward upon the internal spermatic artery.

6. *Obliterated Vaginal Process.*—Referred to previously.

Investments of the Cord.—The constituents of the cord are held together by loose areolar tissue, and they are invested by a fibrous sheath formed by evagination of the structures of the anterior abdominal wall through which the cord passes in its descent; *i.e.*, transversalis fascia (internal spermatic fascia), internal oblique (cremasteric muscle), and the external spermatic fascia from the external oblique fascia.

Ilioinguinal Nerve.—In the inguinal region the ilio-inguinal nerve lies anterior to the internal oblique muscle. By passing through the external abdominal ring it becomes superficial. It is distributed to the scrotum in the male and to the labium majus pudenti in the female, and also to the medial aspect of the upper part of the thigh. It supplies motor fibers to the internal oblique muscle.

Ilio-hypogastric Nerve.—The ilio-hypogastric nerve pierces the internal oblique muscle 1 inch in front of the anterior superior spine, and continues in the inguinal canal beneath the external oblique fascia. It becomes cutaneous by piercing the fascia of the external oblique above the external abdominal ring.

Injury to the above nerves is followed by atrophic and trophic disturbances which predispose to a postoperative recurrence of a hernia. The ilio-inguinal nerve, because of its position and its frequent adherence to the under surface of the external oblique fascia, is often cut. The ilio-hypogastric is higher and above the ilio-inguinal, and for this reason it is less apt to become injured. Inclusion of the nerves in a ligature may result in postoperative neuritis, which may last for months.

CONSTRUCTION OF THE INGUINAL CANAL

A knowledge of the mechanics and the natural forces involved in the construction of the inguinal canal is of utmost importance for a clear understanding of the resistance offered by the structures in the canal to counteract adverse factors in the formation of hernia.

Obliquity of the Canal.—The most important natural obstacle counteracting the formation of a hernia is the obliquity of the inguinal canal. This is nature's attempt to correct a potential defect. Counteraction is brought about by bringing the walls of the inguinal canal closer together whenever there is an increase in intra-abdominal pressure. This is clearly demonstrated in Figure 265. In this diagram, it is found that at the level of the internal ring, adverse pressure is overcome by the internal oblique and the strong aponeurosis of the external oblique. In the middle of the canal, protection is afforded by the fascia of the external oblique anteriorly, and by the transversalis and partly by the conjoined tendon posteriorly. At the external ring, the strong protection of the conjoined tendon and the inguinal ligament is readily manifested.

Walls of the Canal.—The inguinal canal is generally considered to possess: (1) an inlet, the internal abdominal ring; (2) an outlet, the external abdominal ring; (3) an anterior wall; (4) a posterior wall and, (5) a floor. With the usual anatomical conception which follows such description, the canal is often considered, by the student and the surgeon, as an oblong space. The author, however, considers the walls of the inguinal canal similar to a spiral stairway, thereby conforming more closely to the actual anatomical relationship of the various structures under consideration. This is an anatomical truth which can be demonstrated on the cadaver.

In this study we shall not consider the various sides of the canal as separate

units. Instead, each structure, muscle or fascia, will be traced separately to determine the manner in which each winds itself around the cord.

Let us examine the *internal oblique*. The muscle arises from the lateral grooved surface of the inguinal ligament. From this point of origin the fibers arch downward and medially to form the anterior wall of the first part of the inguinal canal.

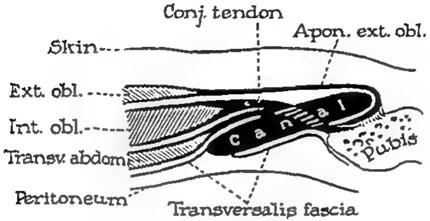

Fig. 265 —Diagram illustrating the obliquity of the inguinal and the natural resistance offered by the walls.

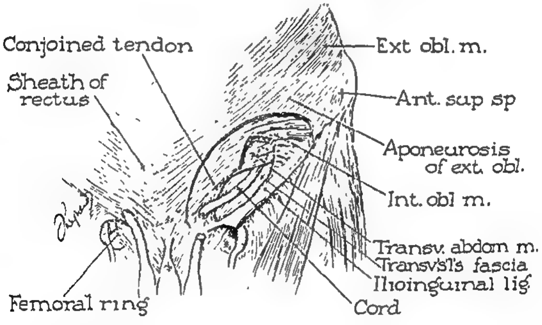

Fig 266 —Diagram to illustrate the relation of the internal oblique to the cord The first part of the cord is covered anteriorly by the fleshy fibers of the muscle. At the middle part the internal oblique is joined by the transversalis to lie medial to the cord. Toward the external ring the fibers lie posteriorly to the cord.

The fibers continue downward and join with the fibers of the transversalis to form the conjoined tendon. At the middle of the canal these fibers arch medially, forming the medial wall (Fig. 266). In the region of the external abdominal ring the conjoined tendon lies posterior to the spermatic cord and gains insertion into the pubic crest and the ilio-pectineal line. In most instances the conjoined tendon is separated from the cord by the lacunar ligament and the reflex inguinal ligament. Hence, the internal oblique at first lies anteriorly, soon becomes the medial wall,

and finally, by blending with the transversalis, forms the posterior wall at the outlet of the canal.

The fascia of the *external oblique* muscle forms the anterior wall throughout the entire extent of the inguinal canal. As the fascia folds upon itself and gains attachment to the anterior superior spine and the pubic tubercle, it affords attachment to the internal oblique and the transverse abdominis muscles in its lateral portion. At the medial end, the inguinal ligament gives rise to the lacunar ligament. This consists of fibers from the opposite side of the external oblique which traverse the linea alba and gain insertion into the front of the pubis and the conjoined tendon. The spermatic cord, as it emerges from the external abdominal ring, lies over the reflex inguinal and the lacunar ligaments, and over the inferior crus of the ring (Fig. 267).

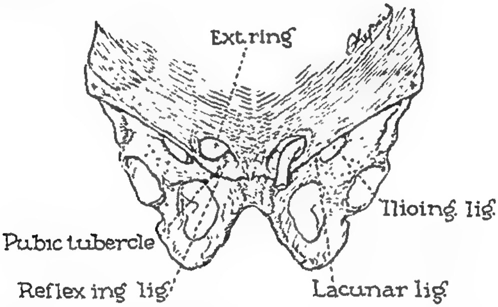

FIG. 267.—Diagram illustrating the connections of the external oblique fascia. Note that the lacunar and the reflex inguinal ligament and the inferior crus lie anterior, whereas, the inguinal ligament in relation to the cord is medial-posteriorly.

It is evident from the above description that the external oblique fascia, inguinal ligament, reflex inguinal ligament, and lacunar ligament, all derived from the same fascial strata, enter into the formation of practically all sides of the inguinal canal.

In the inguinal region the *transverse abdominis* muscle blends with the internal oblique to form the conjoined tendon (also called the *Falx inguinalis*). The fleshy fibers stop just at the internal ring, and from that point they arch downward to gain insertion, through the medium of the conjoined tendon, on the pubis and the ilio-pectineal line. It is of importance to note that whereas the conjoined tendon has an attachment on the ilio-pectineal line of about $\frac{1}{2}$ inch, the transversalis is attached for almost 1 inch. Here again, when the transversalis is traced from the inlet of the inguinal canal, the fibers are found anterior to the cord, whereas close to the outlet they lie posteriorly.

Let us now study the various sections of the inguinal canal. Figure 268 is a sagittal section demonstrating the anterior and posterior walls of the canal. Notice that the fascia of the external oblique forms the anterior wall throughout the entire surface of the canal. It must be observed, however, that in the region of the external

ring the fibers are arched posteriorly, giving rise to the lacunar ligament. The latter is attached to the ilio-pectineal line and is placed anteriorly to the conjoined tendon. In this diagram the relationship of the inguinal ligament to the cord is not shown. The internal oblique in the region of the internal ring, is found anterior to the cord. Soon it arches, slightly medially, and finally blends with the transversalis to form the posterior bed of the canal. The transversalis fascia is demonstrated as forming the deepest layer of the posterior wall of the canal.

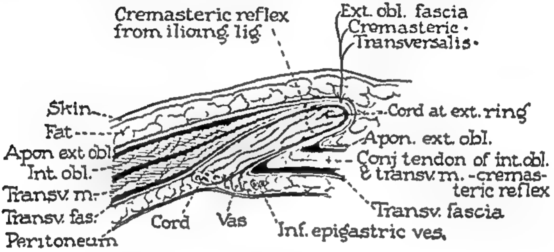

FIG. 268—Obliquity of the inguinal canal.

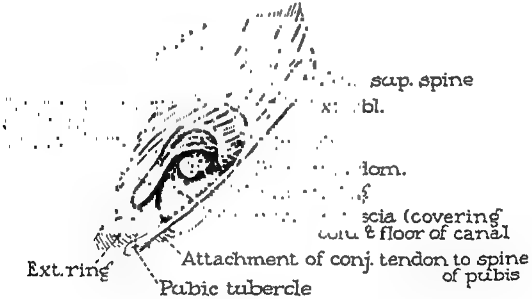

FIG. 269.—Schematic diagram illustrating the spiral stairway construction of the ingunial canal. This section is at the level of the internal ring.

A sagittal section of the canal does not illustrate the spiral effect of these structures. For this reason, the author has attempted, in the following schematic drawings, to show the "spiral stairway" construction of the canal.

Figure 269 is a section of the inguinal canal at the level of the internal abdominal ring. According to McGregor, a true inguinal sphincter exists which is formed by the internal oblique and transversalis muscle. He states that "the sphincter is voluntary in character and normally functions to protect the internal ring opening

of the inguinal canal, first by a constant state of tonus, and second, by voluntary reflex contractions whenever the intraperitoneal pressure is, for any reason, increased." Immediately anterior to the transversalis is the strong fleshy internal oblique muscle and the tense aponeurosis of the external oblique. The spermatic cord is seen lying anterior to the transversalis fascia and beneath the transverse abdominis and the internal oblique muscles. When it reaches the middle of the canal, the cord rests upon the transversalis, with the conjoined tendon posteriormedially, and the external oblique anteriorly (Fig. 270). At this stage the cord has emerged from the constricting sphincter-like action of the internal ring; the internal oblique is now wanting anteriorly, and the transversalis fascia forms its posterior wall.

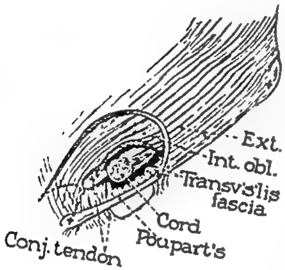

FIG. 270

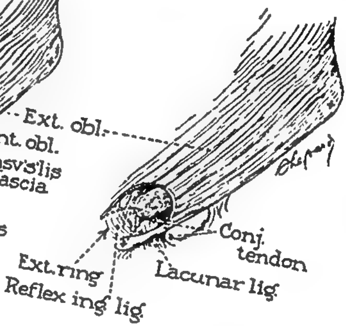

FIG. 271

FIG. 270.—The relationship of the cord in the middle of the canal.

FIG. 271.—The relationship of the cord in the region of the external ring. Note that the cord lies anterior to the conjoined tendon, the lacunar ligament and the inguinal ligament.

Comparing the walls of the canal at these levels, we find that at the level of the internal ring the anterior wall is composed of three layers; transversalis fascia, internal oblique, and external oblique. The transversalis fascia surrounds the cord completely. At the level of the mid-point of the canal, the external oblique forms the antero-lateral border, the internal oblique, the medial border, and the transversalis, the posterior border.

Figure 271 illustrates the construction of the canal in the region of the external ring. Note that the cord lies anterior to the conjoined tendon, the lacunar ligament, and the inguinal ligament. The anterior wall is formed by the external oblique aponeurosis.

From the study just made, one must be impressed by the remarkable protective mechanism which nature has provided in the construction of the inguinal canal. This is especially true in the regions of the outlet and inlet. At the inlet we find

3. Internal spermatic fascia, from the transversalis.
4. Cremasteric muscle, from the internal oblique.
5. External spermatic fascia (intercolumnar fascia), from the aponeurosis of the external oblique.
6. Superficial fascia.
7. Skin.

Direct Hernia.—In a direct hernia the sac pushes ahead the structures which form the floor of Hesselbach's triangle. In medial direct hernia the sac derives a covering from the conjoined tendon as well as from the fascia transversalis. In the medial variety the fat which fills the space of Retzius is commonly seen over the sac. The extraperitoneal fat which covers the sac is often hypertrophied, appearing as a fatty tumor. *From within outward, the coverings of the sac are:*

1. Peritoneum (the sac).
2. Extraperitoneal fat.
3. Transversalis fascia.
4. Conjoined tendon; in the lateral variety the conjoined tendon curves around the medial side of the sac.
5. External spermatic fascia (aponeurosis of the external oblique).
6. Superficial fascia.
7. Skin.

CLASSIFICATION OF INGUINAL HERNIA

Indirect Oblique Hernia.—Indirect hernia may be classified according to the degree of the descent of the sac: (1) in the incomplete type, the sac remains within the inguinal canal; (2) in the complete type, the sac emerges from the inguinal canal at the external abdominal ring; (3) in the scrotal type, the sac passes down into the scrotum. They are also classified according to the degree of patency of the vaginal process, *i. e.*, as congenital or acquired. Clinically they may be classified as reducible, irreducible, inflamed or strangulated.

Congenital Variety.—1. *Complete or Vaginal type.*—This is the result of the failure of the tunica vaginalis to close entirely, thus allowing a hernia to pass into the scrotum (Fig. 274). In this type the protruded viscera come in contact with the testicle. Because the right testicle descends later in life than the left, this variety is found more frequently on the right side.

2. *Incomplete, Funicular Type.*—This is due to partial closure of the funicular process. In this type the funicular process is closed at the epididymis; hence the hernial sac cannot pass into the vaginal process (Fig. 275). It occurs more frequently than the vaginal variety. Often, it is mistaken for the acquired variety. In the vaginal, or funicular type, the sac is firmly adherent to the cord structure, whereas in the acquired variety the sac has a rounded fundus which can be identified and stripped easily from the cord.

3. *Infantile or Encysted Type.*—These are identical terms. This type of hernia was first described by Hey, who called it "infantile," probably because the case he observed occurred in an infant. Later, Copper found this variety of hernia associated with a hydrocele and designated it as an encysted hernia. The latter term is more appropriate because it is anatomical.

In the encysted hernia the funicular process is closed at the internal ring, but remains open below. The hernia passes down behind the opened process (Fig. 276),

or the hernial sac may invaginate into the sac of the hydrocele (Fig. 277). In either case a form of hydrocele is associated with the hernia.

Acquired Inguinal Hernia.—Direct inguinal hernia is usually acquired, and is due to a defective development of the muscles, and conjoined tendon forming the floor of Hesselbach's triangle.

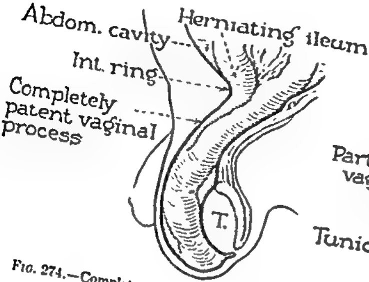

Fig. 274.—Complete congenital hernia.

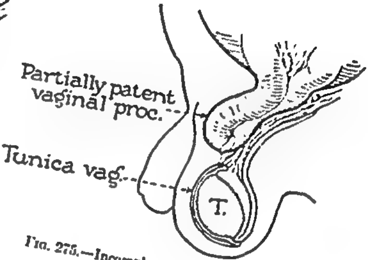

Fig. 275.—Incomplete funicular hernia.

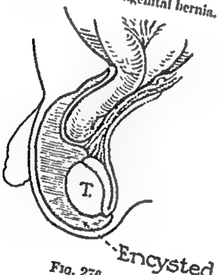

Fig. 276

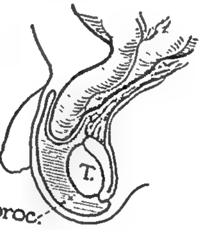

Fig. 277

Fig. 276.—Infantile hernia. The sac passing behind the tunica.
Fig. 277.—Infantile hernia. The sac invaginating the tunica.

In the acquired indirect oblique inguinal hernia, the funicular process is obliterated throughout its entire extent. The hernia takes its origin in the lateral inguinal pouch, and consists entirely of peritoneum which has protruded from within the abdomen. It may extend as far as the head of the epididymis, and if of large size may overlap the testicle.

DIRECT INGUINAL HERNIA

Lateral Direct Inguinal Hernia.—A lateral direct inguinal hernia occurs in the medial inguinal fossa. The latter lies between the inferior deep epigastric artery and the obliterated hypogastric artery (Fig. 278). The medial inguinal fossa is

normally narrow, but deep. It lies behind that part of Hesselbach's triangle which is formed only by the transversalis fascia. This type of hernia is termed "lateral" because it lies laterally in relation to the obliterated hypogastric artery.

Medial Direct Inguinal Hernia.—A direct hernia may escape through the supravesical pouch which lies between the middle umbilical ligament and the obliterated hypogastric artery. This type may also be designated as a supravesical direct hernia. In this variety the urinary bladder is very often the sac.

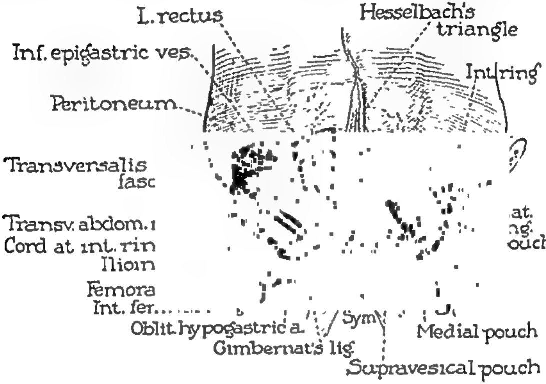

FIG. 278.—The abdominal wall to show the relations of direct hernia in Hesselbach's triangle.

INTERSTITIAL HERNIA

An interstitial hernia is one which develops within an abnormal relation to the anterior abdominal wall. It is often associated with an abnormal descent of the testis. There are three varieties: (1) subcutaneous; (2) interparietal, and (3) intraparietal (Figs. 279 and 280).

Subcutaneous Type.—In this type the sac lies between the subcutaneous tissue and the external oblique fascia. Generally the hernia escapes through the external abdominal ring and travels outward along the inguinal ligament. It resembles a femoral hernia. The diagnosis is simple, since the scrotum is retracted and the testis is absent.

Interparietal Type.—In the interparietal type the sac may be found in one of three different locations: (1) between the transversalis fascia and muscle; (2) between the internal and external oblique muscles; or (3) between the transversalis fascia and the external oblique aponeurosis. Often the testis extends upward with the sac. This is probably due to the closure of the external abdominal ring, so that the testicle and hernial sac can travel only upward underneath the external oblique fascia.

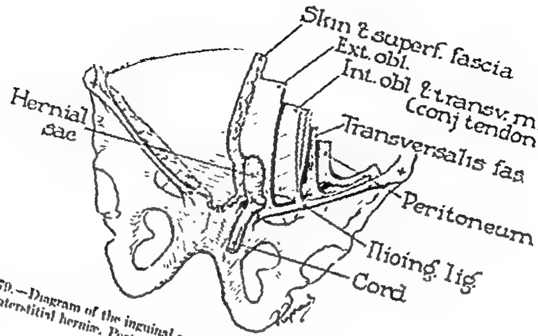

Fig. 279.—Diagram of the inguinal canal illustrating the position of extraparietal, and subcutaneous interstitial hernia. Portions of the different layers of the abdominal wall have been removed.

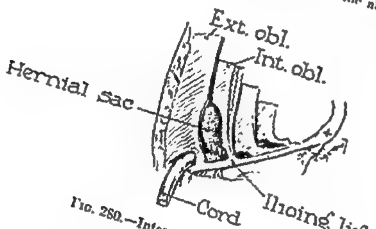

Fig. 280.—Interparietal hernia.

TREATMENT OF INGUINAL HERNIA

In the operative treatment of an inguinal hernia, the following factors must be considered.

Disposition of the Sac.—It is now generally admitted that the removal of the entire sac is an essential step in the operative treatment of inguinal hernia. The importance attached to the treatment of the sac differs among surgeons. The early surgeons did not remove the sac, but closed the opening at the level of the peritoneum and sutured the inguinal rings.

Some surgeons remove the sac high up, leaving no redundant peritoneal pouches, with or without reconstructing the inguinal canal. Soresi advocates a more radical procedure. After splitting the fibers of the external oblique, he separates the internal oblique at a point slightly above the internal ring, exposing the peritoneum. The latter is incised and pulled out so that the internal ring is exposed and exteriorized. The peritoneum is thus fully stretched from the pubis to the internal ring.

The peritoneal opening is then closed. By this method the peritoneum and ring are excluded from the peritoneal cavity.

In the complete congenital hernia, the sac cannot be removed completely, because its fundus is adherent to the testis. In such cases a portion of the sac is left attached to the testis.

Reconstruction of the Inguinal Canal.—Reconstruction of the inguinal canal includes: (1) restoration of the internal ring to its normal position and size; (2) restoration of the normal obliquity of the canal, (3) provision for a strong posterior wall, and (4) strengthening of the anterior wall.

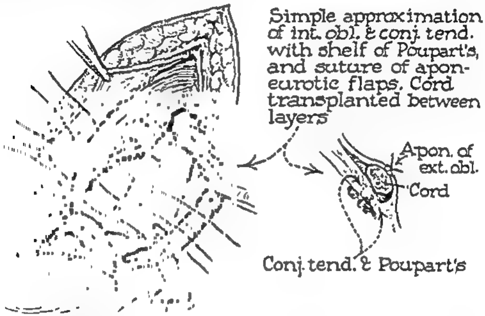

FIG 281.—Bassini operation, illustrating the method of inserting the deep sutures. Inset shows the position of the spermatic cord as it lies in its new bed.

The *classical Bassini operation* consists of suturing the conjoined tendon to the lacunar and inguinal ligaments. In this manner the internal oblique muscle is placed around and below the internal ring, thus a new internal ring is formed, and the cord is displaced (Fig. 281). The objection can be made that the reconstruction is not anatomical, since it disregards the transversalis fascia.

Gregory Connell in 1909, and Pizman in 1921, modified the operation by minimizing and elevating the internal ring by repairing the transversalis fascia below the cord. If the transversalis is greatly attenuated, Connell suggests the utilization of the external oblique fascia, fascial strips, or even the thick-walled sac for suture material.

The principles of *Halsted's operation* consist of suturing the cremasteric muscle and fascia underneath the conjoined tendon and internal oblique muscle. The edge of the conjoined tendon and the edge of the internal oblique are then sutured to the shelving portion of the inguinal ligament. The flaps of the aponeurosis of the external oblique are overlapped. The cord is left undisturbed (Fig. 282).

In the *Ferguson modification* the reconstruction of the canal is made anterior to the cord. The internal ring is narrowed by suturing the transversalis fascia. The conjoined tendon is then sutured to the shelving portion of the inguinal ligament. The aponeurosis of the external oblique is overlapped and sutured.

In the *Andrews modification* of the Halsted method, figure-of-8 sutures are used to unite the internal oblique, the conjoined tendon, and the upper flap of the external oblique fascia to the shelving portion of the inguinal ligament. The lower flap is then lapped over the upper one.

The Andrews modification of the classical Bassini operation consists of suturing the conjoined and the upper flap of the external oblique to the shelving portion of the inguinal ligament underneath the cord. The lower flap is then lapped over the cord and sutured to the upper flap. In this manner the cord is made to lie in a new bed consisting of the aponeurosis of the external oblique. This operation is popular, and for this reason will be described in detail.

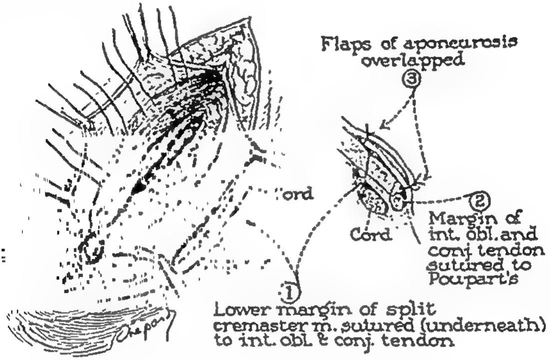

FIG. 282 —Halsted operation.

Treatment of the Spermatic Cord.—The spermatic cord may be dealt with in a variety of ways. In the classical Bassini operation the cord is made to lie upon a new bed consisting of the internal oblique muscle. In the Andrews modification the cord is transplanted and is made to lie between the two layers of the external oblique aponeurosis. The cord may also be placed entirely upon the external oblique fascia, being covered only by the subcutaneous tissue and skin.

In the Halsted and Ferguson modifications where the cord is not transplanted, the reconstruction of the canal is done anterior to the cord. By leaving the cord alone, postoperative complications to the spermatic cord and the testicle are eliminated.

The question whether or not to transplant the cord has received considerable attention. Some surgeons transplant the cord as a matter of routine, while others do this only for direct hernia. There are still others who never transplant the cord, regardless of the type of hernia. From an anatomical point of view it is best to transplant the cord in direct hernia. In this case the defect is in the floor and at the apex of Hesselbach's triangle; hence, the object of the operation should be to reinforce the floor behind the cord. In the indirect type of hernia, the best

results are obtained when the natural relationship of the cord to the canal and the normal obliquity are maintained. This is obtained by strengthening the anterior and posterior walls without displacing the cord.

OPERATIVE TECHNIQUE

Skin Incision.—Before making the skin incision palpate the pubic tubercle, the anterior superior spine, and visualize the topographical anatomy (Fig. 283). Note that the internal ring is located 1 centimeter medial to the mid-point (femoral point) of the inguinal ligament. The spermatic cord can be felt emerging through the external abdominal ring immediately above the pubic spine. The inferior epigastric artery may be outlined by a line drawn from the femoral point to the umbilicus. The artery, together with the inferior part of the external border of the rectus and the medial third of the inguinal ligament, bounds the Hesselbach's triangle.

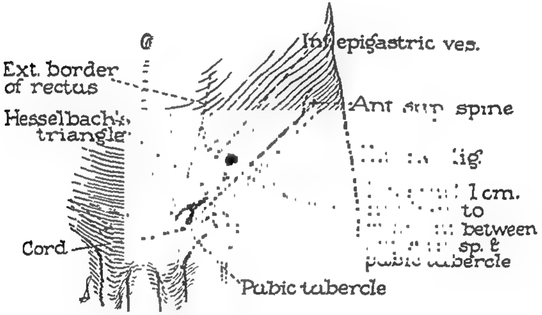

FIG. 283.—Topographical anatomy of the inguinal region.

The skin incision is made approximately $\frac{3}{4}$ inch medial and parallel to the inguinal ligament, beginning just beyond the internal ring and extending to the pubic tubercle (Fig. 284). If a previous operation has been done, the old scar is excised. The incision is carried down through the subcutaneous tissue, exposing the aponeurosis of the external oblique. When cutting through the subcutaneous tissue, watch for the superficial epigastric and the circumflex iliac vessels. If possible, isolate, clamp, and ligate them before dividing. Perfect hemostasis is essential in order to identify the tissue adequately and to avoid infection.

Splitting of the External Oblique Aponeurosis.—Generally the thinnest part of the external oblique fascia is found directly over the inguinal canal. When the fibers are widely separated, the incision in the aponeurosis should be made in the direction of the split fibers. To avoid injuring the ilio-inguinal nerve a groove director is applied underneath the fascia, extending from the inner side of tht external ring to just beyond the internal ring (Fig. 285). With the groove director as a guide, the aponeurosis is incised with either a scalpel or scissors. The inner

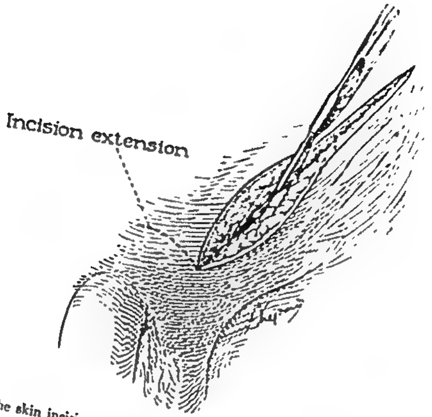

FIG. 284.—The skin incision. Dotted line represents an extra incision for the exposure of the rectus fascia.

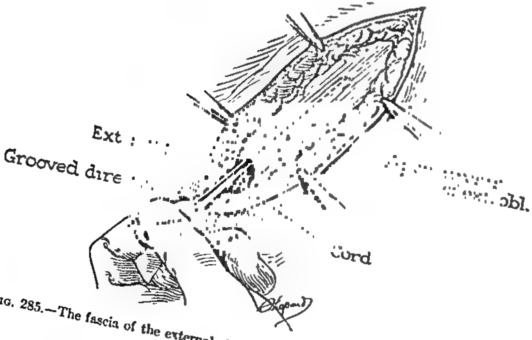

FIG. 285.—The fascia of the external oblique is split in the direction of the fibers over a groove director.

surface of the external oblique (inguinal ligament) is now grasped with Allis forceps. It is then freed with gauze dissection of all fat and areolar tissue until the shelving portion of the inguinal ligament is exposed completely from the internal ring to the pubic tubercle. The outer surface of the external oblique fascia is separated for a distance of ¼ inch from the underlying internal oblique muscle. This procedure makes the subsequent closure of the canal easier.

Every precaution should be used to avoid injury to the ilio-hypogastric and the ilio-inguinal nerves. The latter is very often cut because of its position and because it is often adherent to the under surface of the external oblique fascia. The ilio-hypogastric is higher and above the ilio-inguinal; hence it is less apt to become injured. The most common methods of injuring these nerves are:

Fig 286.—Freeing the sac from the cord. Preferably done with sharp dissection.

1. Cutting the nerves when incising the external oblique fascia, because the surgeon has failed to identify them.
2. Forcible scraping the internal oblique, and tearing of the nerve while exposing the inguinal ligament.
3. Picking up the nerves with forceps and carrying them "out of harm's way"—this tears away their branches.
4. Including the nerve in a suture.

Mobilization of the Cord and Sac.—After the fascia of the external oblique has been incised and the inguinal ligament has been freed, the following structures are seen: (1) the shelving portion of the inguinal ligament; (2) the lacunar ligament; (3) the conjoined tendon; (4) the internal oblique muscle; and (5) the spermatic cord with the hernial sac.

Beginning at the pubic tubercle, the spermatic cord is lifted from its bed as far down as the internal ring. This is done cautiously to avoid injuring the blood vessels, the areolar tissue around the cord, and the arched fibers of the conjoined tendon. The cord, should be stripped cleanly from the floor of the canal.

The cremasteric muscle and the underlying fascial structures are now incised to expose the sac. The latter is recognized by its translucent bluish or lead color, and the ease in which it slides between the fingers. If possible the sac should be opened and its contents reduced *before* isolating it. Adhesions between the sac and the contents are carefully dissected to avoid injury to the omentum or to the intestine. If the contents of the sac consist of a large and irreducible mass of omentum, or if the omentum is strangulated, it is amputated and ligated in small pedicles. Make certain that all bleeding points are ligated to prevent subsequent intra-abdominal hemorrhage.

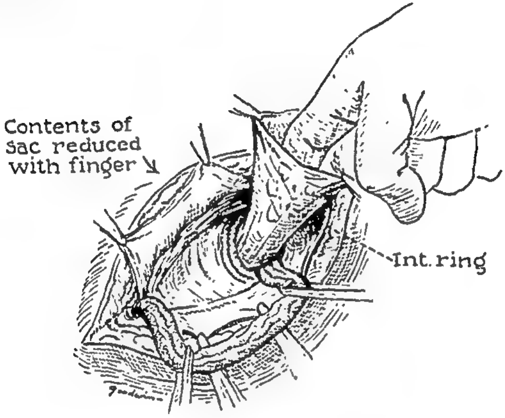

FIG. 287.—The sac completely free and the contents being reduced.

Large scrotal hernia containing numerous loops of bowel may offer some difficulty in the reduction of the intestine. Figure 288 is the author's method of reducing the intestine without causing any injury to the bowel. A loop of bowel is sought which is seen to lead into the abdomen and by gentle pressure with finger or gauze-covered forceps it is pushed into the abdomen. The bowel is "fed" into the abdominal cavity, following the same loop, until the sac is completely reduced. It is found that after a few inches of the bowel has been returned into the abdomen the remainder slides back much easier.

After the sac has been emptied it is separated from the cord up to and including the internal ring well inside of the parietal peritoneum. In order to avoid injuring the vas while separating the sac, the author uses a Cameron right angle surgilite to transilluminate the sac and cord. In this manner, with the surgilite placed against the cord, the dissection of the sac is accomplished with a minimum of trauma or danger to the vas.

Now introduce a finger through the neck of the sac and determine the size and

position of internal ring, and also the strength of the posterior wall of Hesselbach's triangle. Instead of using the finger, Cameron right angle surgilite may be introduced through the neck of the sac into the abdominal cavity with the tip of the surgilite directed toward the pubic tubercle. Figure 289 demonstrates the surgilite transluminating the apex of Hesselbach's triangle. If a direct hernia is present, the sac will be transilluminated regardless of its size (Fig. 290). If the sac is not present, but the area is transilluminated freely, it indicates that the floor of the

Fig. 288.—Author's method of reducing an incarcerated hernia. A loop of bowel is sought which leads into the abdomen and by gentle pressure it is pushed into the abdomen with finger or hemostat.

triangle is weakened as a result of a displaced or thinned-out conjoined tendon. The amount of resistance offered by the tissues also helps to determine the strength of these structures.

Too much emphasis cannot be placed upon the advisability of searching for a hernia at the apex of Hesselbach's triangle. The likelihood of recurrence is greatly diminished if, upon examination, a sac is found and extirpated.

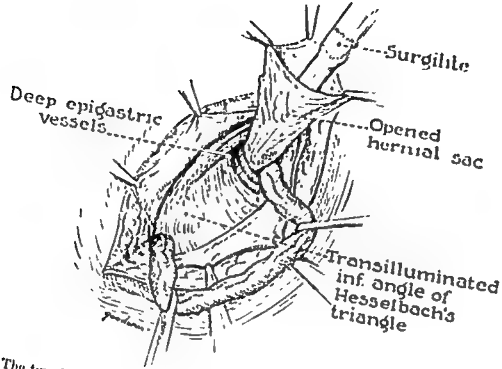

FIG. 289.—The tip of Cameron surgilite transilluminating the apex of Hesselbach's triangle.

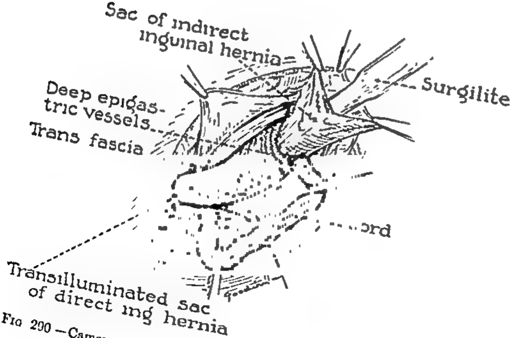

FIG 290 —Cameron surgilite transilluminating a coexisting direct hernia.

Extirpation of the Sac.—After determining the condition at the floor of the canal, the next procedure is the amputation of the sac. We attach considerable importance to the first step, which consists of pulling the sac downward until the peritoneum around the internal ring is well drawn out. The sac is then twisted as illustrated in Figure 291, *a*. When twisting the sac, make certain that no viscera or omenta are included. While traction is made on the twisted sac, a crushing forceps is applied at its base. The base is then transfixed with a suture just below the groove

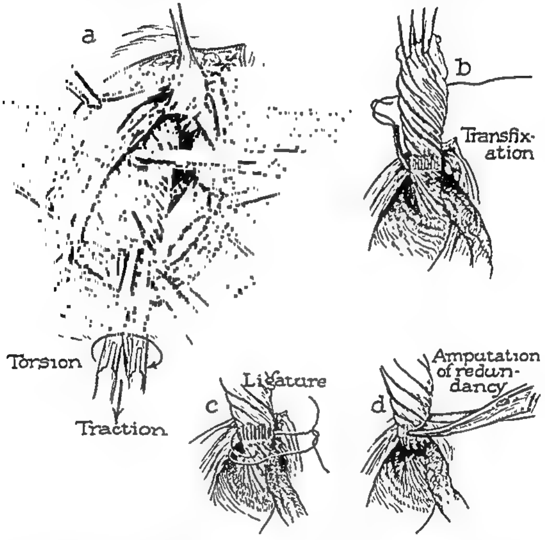

FIG 291.—Extirpation of the sac: *a* traction and torsion of the sac; *b* transfixation of the sac; *c* ligation; *d* removal of the redundant portion of the sac above the transfixed ligature.

formed by the forceps (Fig. 291, *b*). The redundant portion of the sac is then cut away distal to the ligature. By this method the ligated stump will retract about 1 to [illegible] inches above the normal position of the internal ring.

If the neck of the sac is wide, a purse string suture is applied and the redundant portion extirpated. The ends of the suture are left long, and are threaded on a curved needle which is then introduced underneath the internal oblique muscle and brought out 1 inch from the internal ring. The ends of the suture are then tied upon the surface of the external oblique. This procedure also draws on the peritoneum, thereby elevating the internal ring away from its original position.

The sac of a direct hernia is located medially to the inferior epigastric artery; hence it occupies the lower half of the inguinal canal. In this type of hernia the sac has a wide base and is sausage-shaped. For this reason, it is difficult to transfix, amputate, and transplant the stump of the sac, as was done for the indirect hernia. If the sac is small, it is not essential to open it, except to detach adherent contents. In a large hernia the sac should be opened, the contents reduced, and the excess portion cut off. The closure is then made in the same manner as in suturing the

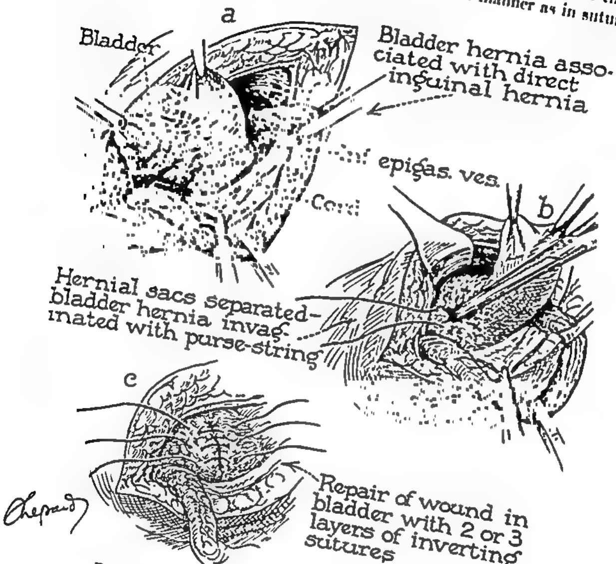

FIG. 292.—Bladder hernia associated with direct hernia.

peritoneum after an abdominal operation. If the sac has a neck, it may be treated in the same fashion as an indirect hernia.

When a direct hernia is found coexisting with an indirect hernia, the two sacs are converted into one. This is accomplished by first isolating and opening the indirect sac, and then making traction on it until the direct sac is drawn through the internal ring, thus converting the two sacs into one. The combined sac is then treated similarly as in the sac of an indirect hernia.

When operating for direct hernia, the surgeon should use every precaution to avoid injuring the urinary bladder. It is recognized by the yellowish pre-hernial

fat, by the deeper color, and by the increased number of small blood vessels on its surface. The bladder should be carefully isolated and returned into the abdomen, and the wound closed with a purse string suture using chromic catgut No. 0. Figure 292 illustrates a method of treating the urinary bladder associated with direct hernia. If the bladder is accidentally opened, clamp the wound and invert its edges with two layers of interrupted Lembert stitches, using chromic catgut No. 0. These stitches must not penetrate the mucosa.

Reconstruction of the Inguinal Canal.—The first consideration in the reconstruction of the inguinal canal is the repair of the internal ring. The ultimate objective is to elevate and minimize the ring by suturing the transversalis fascia below the cord. This may be accomplished according to the methods of Connell, Pizman, or Seelig.

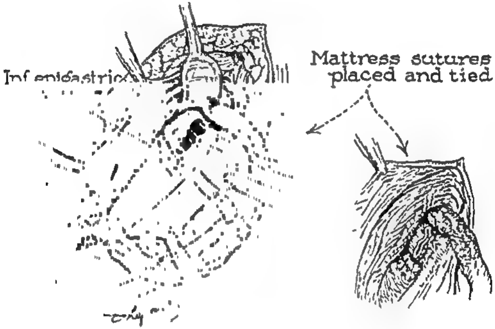

FIG. 293.—Connell method of repairing the internal ring. Mattress sutures are applied to plicate the transversalis fascia below the cord.

As early as 1908, Connell realized the importance of reconstructing the internal ring and the posterior wall of the canal by suturing the transversalis fascia below the cord. As illustrated in Figure 293, Connell applies mattress sutures to plicate the transversalis fascia beneath the cord. Generally two of these stitches are sufficient. If the transversalis is attenuated, the thick wall sac may be utilized as suture material.

Seelig introduces a finger in the neck of the sac and stitches the transversalis fascia throughout the entire extent of the posterior wall (Fig. 294). The characteristic step of Pizman consists in suturing the peritoneum and fascia transversalis to the shelving portion of the inguinal ligament (Fig. 295). Regardless of which method is employed, the surgeon must exercise care to avoid injury to the inferior epigastric artery and to the external iliac vessels. The advantage of using any one of these methods is that the internal ring will be elevated to its normal position behind the internal oblique muscle with a firm lining of fascia around it.

The next step consists of suturing the conjoined tendon and the fascia of the external oblique to the shelving portion of the inguinal ligament. This is best

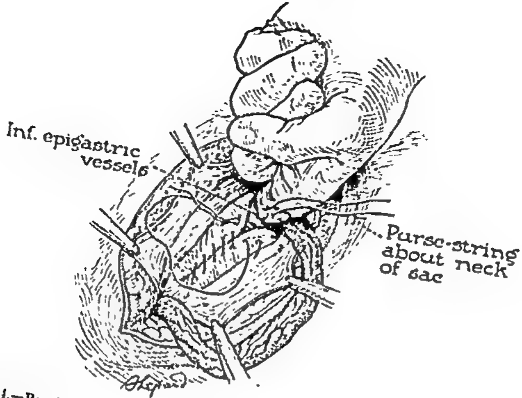

FIG. 294.—Repair of the internal ring according to the method of Seelig. The index finger has been inserted into the neck of the sac to protect abdominal viscera when suturing the transversalis fascia.

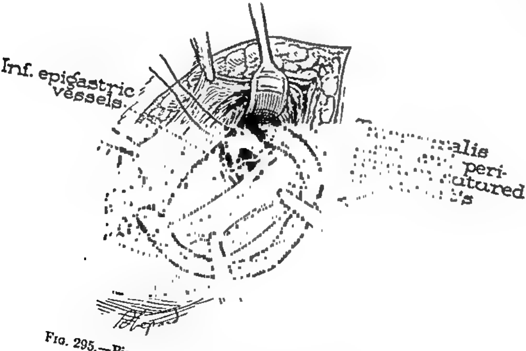

FIG. 295.—Pizman's method of repairing the internal ring

carried out according to the method advocated by E. Wyllys Andrews. The Andrew stitch (Fig. 296) is applied in the following manner. With the aponeurotic flap of the inguinal ligament held perpendicularly, the needle is passed from without, entering the canal posterior to the shelving portion of the inguinal ligament. The needle is then passed underneath the cord and a bite is taken into the conjoined tendon. While inserting the suture the posterior to the shelving edge, care must be exercised not to puncture the external iliac vessels. To avoid this complication, the author places the index finger of the left hand underneath the shelving edge in order to guide the point of the needle away from the underlying vessels (Fig. 297).

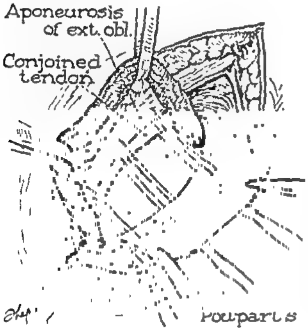

FIG. 296

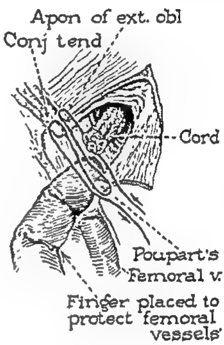

FIG. 297

FIG. 296 —Three Andrews stitches have been inserted.

FIG. 297.—Cross-section to illustrate the Andrews stitch and method of protecting the underlying vessels by placing the index finger beneath the shelving portion of the inguinal ligament.

After the conjoined tendon has been grasped, the stitch is reversed and a bite is taken into the shelving edge of the inguinal ligament. The needle is reversed again, and the upper flap of the aponeurosis of the external oblique is caught. Next, the needle is passed through the inguinal ligament above the shelving edge, at a point close to the one of entrance (Fig. 296).

Three or four similar sutures are inserted, and none is tied until all are in place. The lowermost suture is applied close to the pubis, whereas the uppermost one is placed close to the internal ring. The latter stitch need not be too close, if the transversalis has been sutured according to the method of either Connell, Seelig, or Pizman. When tying the suture close to the internal ring, leave enough room for the escape of the cord. When all sutures are tied, the conjoined tendon and the aponeurosis of the external oblique will be brought together in apposition to the shelving edge of the inguinal ligament. The stitches are tied outside of the canal, and the tying is much easier. Avoid tying the sutures too tightly, because they cut

through or strangulate the tissues. If necessary interrupted sutures may be applied to reinforce the Andrews stitches.

Next, the cord is placed on its new bed and the aponeurotic flap of the inguinal ligament is sutured over the cord to the external oblique fascia. As illustrated in Figure 298, one or two mattress sutures may be applied to imbricate the flaps of the external oblique above the ring. If the ring is found to be greatly widened, the sutures should include the internal oblique muscle.

The flap of the external oblique fascia is now overlapped and sutured with a continuous chromic catgut No. 0 (Fig. 300). The operation is completed by suturing the superficial fascia with interrupted plain catgut No. 00.

Before the incision is closed, see that the cord is not constricted, that the vascular supply to the testis is preserved, and that all bleeding points are controlled. Observance of these precautionary measures will prevent the possibility of testicular atrophy, hydrocele, or both.

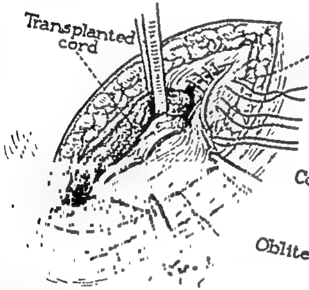

Fig. 298

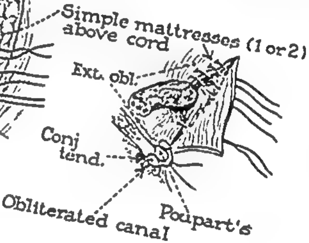

Fig. 299

Fig. 298 —Andrews stitches have been tied and two mattress sutures have been applied to imbricate the external oblique fascia above the internal ring.

Fig. 299 —Diagram illustrating the reinforced internal ring.

This operation provides a new floor behind the cord composed of the conjoined tendon and the fascia of the external oblique (Fig. 301). The internal ring has been elevated to its normal position behind the internal oblique muscle, thus bringing the ring to its normal position. This technique, combined with high ligation of the sac, offer the best protection against recurrence of the hernia.

VARIATIONS IN TECHNIQUE

Hernia in Infants and Children.—It has been estimated that nearly 50 per cent of infants have a patent process vaginalis at birth, hence, hernia is most frequent during the first year of life. During the next five years spontaneous cure occurs in the great majority of these. This is due to closure of the process vaginalis. Direct hernia is extremely rare in infants. Over 95 per cent of hernia in infants are reducible. The content in the sac is usually small intestine or omentum; rarely

other viscera are found. Operative treatment offers the best results and is indicated in any of the following conditions: (1) when the hernia is irreducible or strangulated. In these cases operative interference is imperative; (2) when conservative treatment has failed to bring about cure; (3) when the wearing of the truss is impractical or impossible; (4) a hydrocele associated with a hernia; (5) when the testicle is in the inguinal canal; and (6) when adherent omentum or inflammatory changes causes pain. Operative treatment may be carried out safely at any age; usually after the fourth week.

In infants and in children with recent hernia, radical operation such as is done for the adult is seldom indicated. As a rule, there is no great destruction of the structures of the inguinal canal, hence high ligation of the sac without suturing of the fascia and muscles will suffice. Additional safety may be obtained by suturing

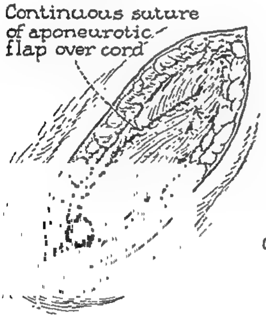

FIG. 300

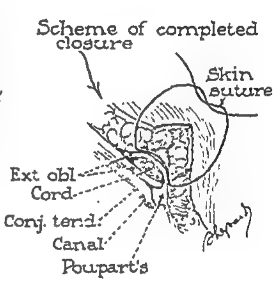

FIG. 301

FIG. 300 —The inguinal flap has been sutured over the cord to the fascia of the external oblique.

FIG. 301 —Diagram illustrating the relationship of the cord in its new bed

the fascia transversalis medial to the internal ring. In other instances—for example, when the hernia is of extremely large size, in which case there is a large internal ring and the obliquity of the canal is destroyed, the procedure should be the same as in the adult.

Sliding Hernia.—Occasionally the cecum (in the right inguinal hernia) or the sigmoid (in the left inguinal hernia) is found in the sac. Such a hernia is known as a "sliding hernia," providing the posterior surface of the bowel is covered incompletely with peritoneum (Fig. 302, *b*). In these cases the hernial sac must be opened with extreme care to avoid cutting into the bowel. The incision into the sac is made on the anterior surface (Fig. 302, *a*) and widely opened to the internal ring. The bowel is then freed carefully and an incision is made in the ower flap of the sac as illustrated in Figure 302, *c*. The edges of this incision are then sutured over the posterior surface of the bowel (Fig. 302, *e*). The bowel is inverted through the hernial ring (Fig. 302, *f*) and a purse string suture completes the closure of the ring.

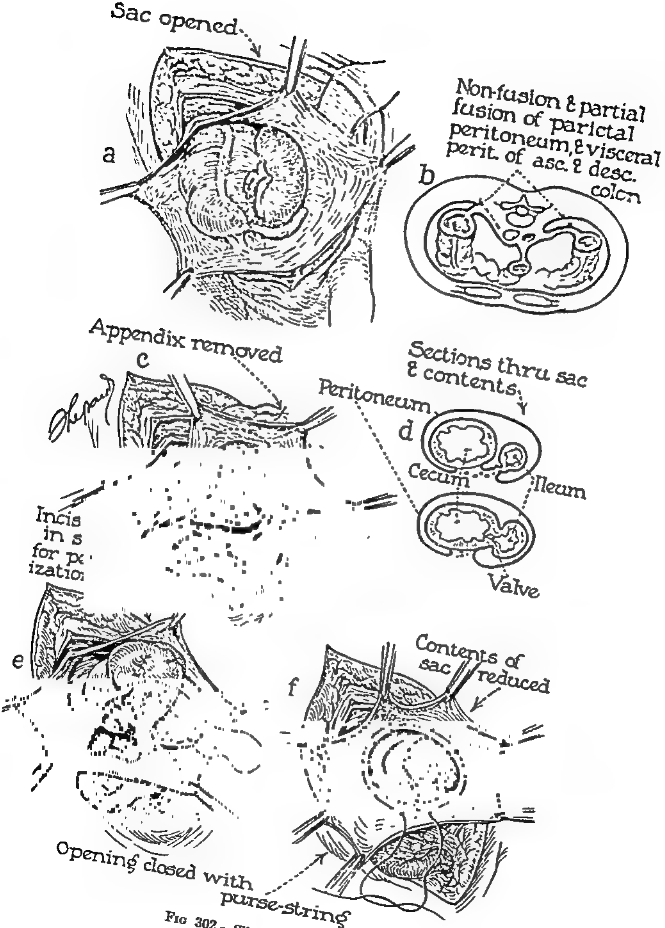

FIG 302.—Sliding hernia.

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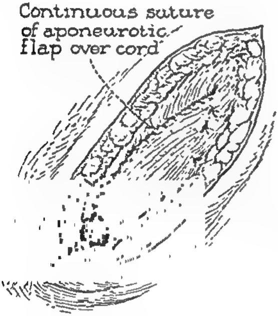
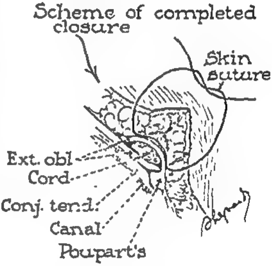


Fig. 300



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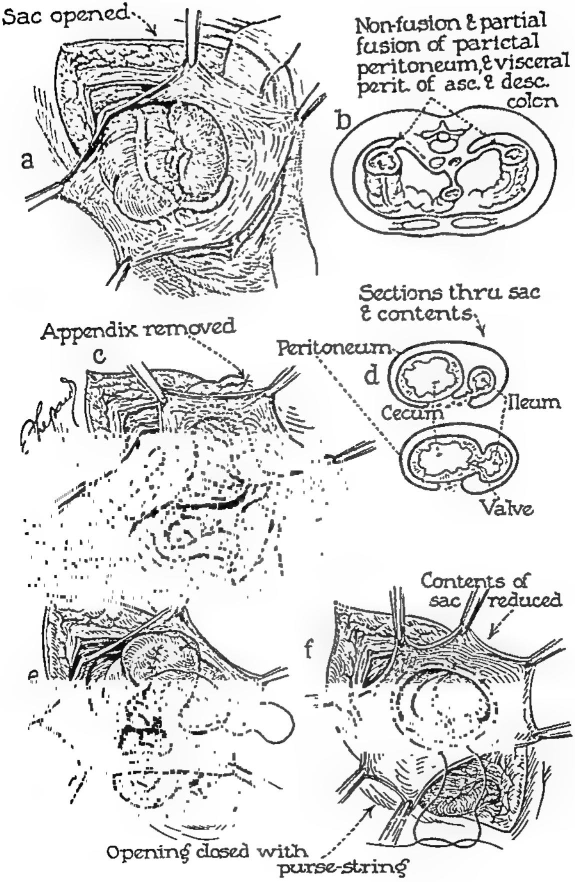

FIG. 302.—Sliding hernia.

The remaining portion of the sac may be trimmed off. The operation is completed as described for inguinal hernia.

Richter's Hernia.—Richter's hernia is a strangulated hernia in which only a part of the circumference of the intestine is caught in the constricting ring. Richter described this type of hernia in 1785. Occasionally it is incorrectly called Littre's hernia, however, this name should be reserved for a hernia containing Meckel's diverticulum. Other synonyms applied to Richter's hernia are: Lavater's hernia, pinched hernia, partial enterocele, and masked hernia.

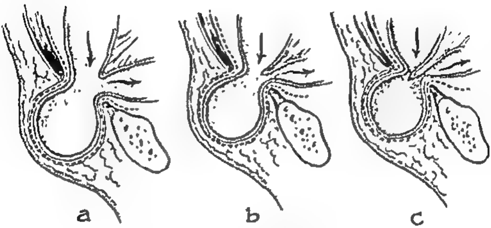

Fig. 303.—Richter's hernia. The degree of strangulation depends upon the amount of the convex surface of the bowel within the sac.

The ileum is most often the strangulated bowel. It is most frequently found in femoral, obturator, and inguinal herniæ. The author has had a case in which the strangulation occurred in an incisional hernia. The physical signs and symptoms of Richter's hernia are the same as those of a complete strangulation with obstruction of the small bowel, except that in partial enterocele the strangulation, since it is not complete permits intermittent passage of gas and intestinal contents. Hence, in the incomplete type the two cardinal symptoms of obstruction, vomiting and abdominal distention may be absent.

Figure 303 illustrates the various degrees of strangulation within a hernial sac and the obvious consequences can be readily deduced. In Figure 303, *a*, only a small portion of the convex surface of the intestinal loop is involved, hence permitting free passage, while in "*b*" the lumen is practically encroached upon. Local signs are often absent, or if present they are improperly interpreted; thus delaying the operation and inviting gangrene of the constricted portion of the bowel. Perforation of the bowel may occur into the sac in a grossly mismanaged case.

Early operation is of utmost importance. Every case has some suggestive symptom which should lead one to suspect the true condition. To wait for all of the symptoms to unfold is to invite disaster. Procrastination is most often due to assuming that a small, slightly tender tumor in the inguinal or femoral region is an inflamed gland, especially when the condition presents local signs of inflammation and obstructive bowel symptoms are absent. In these cases it is logical and good surgical sense to incise an inflamed lymph gland rather than to wait and thus miss an early strangulated bowel.

The operative technique is the same as in any other strangulated bowel. If the segment is viable it may be returned into the abdomen. If there is some

question as to its viability the segment may be replaced into the abdominal cavity for five minutes and then inspected. If gangrenous, the segment is resected and the two ends anastomosed. For Richter's hernia of the femoral, obturator or sciatic regions, the abdominal route is advised.

Littre's Hernia.—Littre's hernia is reserved for a hernia in which Meckel's diverticulum is strangulated. This type of hernia is extremely rare. Weinstein made a survey of the literature in 1938 and found that there were 32 instances in which Meckel's diverticulum was found in a femoral hernia. Of these, 21 were accepted as strangulated femoral herniæ of Meckel's diverticulum. Weinstein did not give the incidence of this condition in inguinal herniæ. However, it may be assumed that this type occurs more often in inguinal hernia. Treatment consists of excision of the diverticulum and the repair of the hernia in the usual manner.

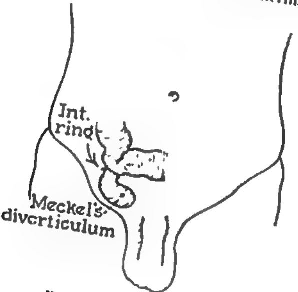

FIG. 304.—Littre's hernia.

Inguinal Hernia in the Female.—Herniorrhaphy is much easier to perform in women than in men, because there is no important structure within the inguinal canal to prevent complete closure. The inguinal canal in the female contains the round ligament, the genital branch of the genitocrural nerve, and the sac. The sac corresponds to the process vaginalis in man and was first described by Nuck in 1672; hence, the term, Canal of Nuck. The same technique as described in this chapter may be employed to repair the hernia. If necessary, the round ligament may be utilized as suture material.

Fascial Sutures.—In some cases, when more effective union is desired, the method of McArthur or that of Gallie is of value. Both use living fascia as a suture. The McArthur technique consists of cutting a narrow strip of fascia from the medial flap of aponeurosis of the external oblique. This is threaded on a needle and used to suture the conjoined tendon to the shelving edge of the inguinal ligament. Another strip may be used to suture the fascia of the external oblique to the shelving edge. However, this is not necessary, since the fascia of like structure heal very well when sutured with catgut, cotton, or silk.

The Gallie technique makes use of living sutures derived from the fascialata of the thigh. This method is of value in a recurrent direct hernia. An incision is made on the outer surface of the thigh, almost as long as the sutures to be removed.

Because of the extra time required to secure this strip of fascia, the assistant can secure the fascia while the surgeon performs the operation. Masson utilizes a fascial stripper for obtaining the fascialata (Fig. 305). The instrument consists of two tubes of steel. The inner tube is slightly longer than the outer one and has an opening in one side, close to the lower end, through which the upper end of a strip of fascia is threaded. The outer tube, which has a cutting edge on its lower end, is used to cut the fascia free. This instrument saves considerable time. After the strip of fascia has been obtained, it can be subdivided into three or four separate sutures. While one is being used, the others can be retained within the incision in the thigh.

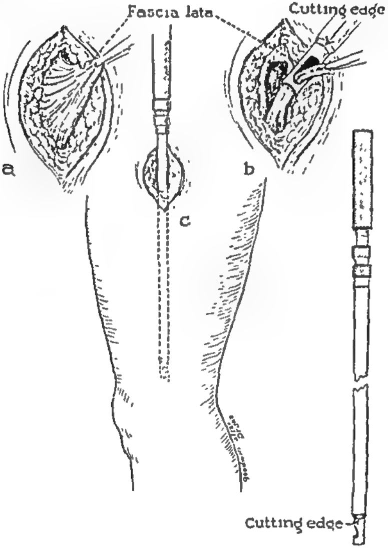

Fig. 305 —Masson stripper used for obtaining fascialata in the repair of hernia.

QUESTIONNAIRE

1. Discuss the process of the descent of the testis.
2. What is the role of the gubernaculum?
3. How is the tunica vaginalis formed?
4. What is the ligamentum vaginalis?
5. What may result from partial or non-obliteration of the vaginal process?
6. [illegible] n the female?
7. [illegible] the internal inguinal ring.
8. [illegible]

9. Does the internal ring possess a sphincteric action? Explain.
10. Give the anatomy of the external abdominal ring.
11. Explain why the external abdominal "ring" is triangular in shape.
12. What is the name given to the layer of fascia of the external oblique that is prolonged downward over the spermatic cord?
13. What is the purpose of the interlacing fibers at the external ring?
14. Give the contents of the inguinal canal.
15. What is the composition of the spermatic cord?
16. Describe the coverings of the spermatic cord.
17. What important factor counteracts the formation of hernia?
18. Explain the role of intra-abdominal pressure and the obliquity of the inguinal canal in herniæ.
19. The inguinal canal is generally considered to possess what walls?
20. Explain the "spiral stairway" construction of the inguinal canal.
21. Give the relationship of the cord at the following levels: (a) internal ring; (b) middle of the canal; (c) external ring.
22. Give the boundaries of Hesselbach's triangle.
23. How are the medial inguinal pouches and the supravesicular pouches formed?
24. What is the name given to a hernia escaping through Hess... ch's triangle?
25. What is a lateral direct hernia?
26. What is a medial direct hernia?
27. Discu...
28. Wh... hernia.
29. Giv... es between a direct and an indirect hernia.
30. Give ... verings of the sac of direct and indirect herniæ.
31. Classify indirect inguinal hernia.
32. What is the name given to a hernia which is due to failure of the tunica vaginalis to close entirely?
33. Why is this variety most frequent on the right side?
34. What is the name given to a hernia due to partial closure of the tunica vaginalis?
35. This type is often mistaken for the acquired type—how can they be distinguished during the operation?
36. Define: encysted hernia.
37. Explain the origin of the acquired indirect hernia.
38. Define: interstitial hernia.
39. Which type of interstitial hernia resembles a femoral hernia.
40. Give the essential steps for the operative cure of hernia.
41. Why is the removal of the sac an essential procedure?
42. What are the essential features of the classical Bassini operation?
43. What is the objection to the Bassini operation?
44. Give the essential features of the Halsted and Ferguson operations.
45. Describe the skin incision for inguinal herniorrhaphy.
46. What care must be taken when making the incision?
47. Why should the incision of the external oblique fascia be made in the direction of its widely separated fibers?
48. What nerves must be avoided?
49. How would you mobilize the cord?
50. How would you identify a coexisting direct hernia?
51. Discuss the treatment of the hernial sac.
52. How is the sac treated in a direct hernia?
53. When a direct hernia is coexisting with an indirect hernia, how would you treat the two sacs?
54. What is meant by "minimizing and elevating" the internal ring?
55. Describe three methods of treating the internal ring.
56. What are the essential characteristics of the Andrews operation?
57. Describe the technique of the Andrews operation.
58. How can you avoid injuring the external iliac vessels during the operation?
59. What is the location of the cord after the Andrews operation?
60. What precautions are used to prevent testicular atrophy or hydrocele?

61. Discuss the repair of hernia in children.
62. Give the indications for herniorrhaphy in infants and children.
63. Define: sliding hernia.
64. [illegible]
65. [illegible]
66. [illegible]
67. Discuss the symptomatology of Richter's hernia.
68. Define: Littre's hernia.
69. Discuss the treatment of inguinal hernia in the female.
70. Give the essential features of McArthur's fascial suture technique.
71. Give the essential features of Gallie's fascial suture technique.

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CHAPTER 45

FEMORAL HERNIA

By A. V. Partipilo

ANATOMY

Femoral Triangle.—Successful repair of a femoral hernia requires complete understanding of the surgical anatomy of the femoral and inguinal regions. The anatomy of the inguinal region has been covered in the previous chapter. The anatomy of the femoral region, although relatively simpler, is often poorly understood.

The femoral trigone or Scarpa's triangle is a superficial area on the upper part of the thigh. It is bounded above by the inguinal ligament (base), laterally by the medial border of the Sartorius muscle, and medially by the adductor longus muscle

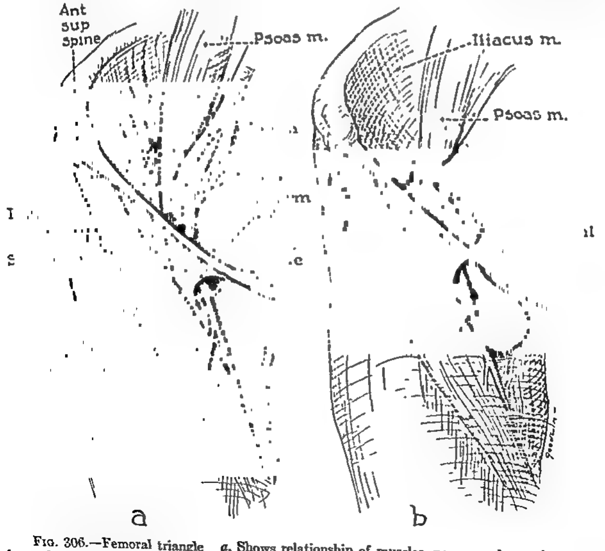


Fig. 306.—Femoral triangle *a*, Shows relationship of muscles, nerves and vessels of the femoral triangle; *b*, illustrates the anatomical relationships of a hernial sac as it descends into the fossa ovalis.

(Fig. 306, *a*). The floor of the trigone is composed of a muscular bed with the iliacus situated laterally, the psoas intermediate, and the pectineus medially. The femoral vessels lie on this muscular bed and are separated from the muscles by a layer of fascia which is part of the femoral sheath. The roof of the triangle is formed by the continuation downward of the transversalis fascia.

The femoral sheath is a conical, membranous investment of the femoral vessels. It is derived from and is a continuation of the fascial lining of the abdominal cavity. The layer of fascia in front of the vessels is a continuation of the fascia transversalis while the layer passing behind is a continuation of the iliac fascia. The sheath is about 1½ inches in length, and is divided into three compartments: (1) a lateral

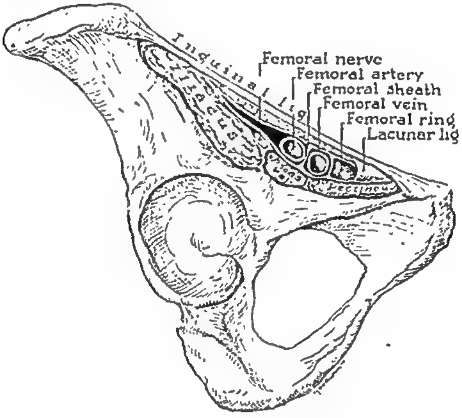


Fig 307.—The femoral sheath.

compartment for the femoral artery and the lumbo-inguinal branch of the genito-femoral nerve; (2) an intermediate compartment for the femoral vein; and (3) a mesial compartment for the femoral canal. Fascial bands or septæ separate the three compartments. The femoral nerve is found external to the femoral sheath in the groove between the psoas and the iliacus muscles (Fig. 307). As illustrated in Figure 306, *b*, the femoral vein is in close proximity to the femoral canal and as the hernial sac descends into the canal it comes in contact with the terminal portion of the long saphenous vein which enters the femoral vein through the fossa ovalis.

Femoral Canal.—The femoral canal is the passage through which a hernial sac enters the thigh. It is about ½ inch long, and contains lymphatic vessels and fat. Normally it is obliterated and cannot be demonstrated unless the femoral sheath is separated from the femoral vein. The proximal end of the canal is the femoral ring which communicates with the abdomen. The ring is bounded anteriorly by the

inguinal ligament, posteriorly by the origin of the pectineus fascia from the pubis, medially by the lacunar ligament, and laterally by the femoral vein (Fig. 307). It is oval in shape, and its width is greater than its length. It is larger in the female (¾ to 1 inch wide) because the lacunar (Gimbernat's) ligament is narrower, weaker, and less securely attached in females than in males (Watson).

Fossa Ovalis.—A femoral hernia after passing through the femoral ring, enters the femoral canal and is directed downward to the fossa ovalis. The fossa ovalis is an oval opening in the fascialata of the thigh permitting the passage of the long saphenous vein into the femoral vein. The center of the fossa is situated 1½ inches distal and lateral to the pubic tubercle. The fossa is covered by the fascia cribosa, a deep layer of the superficial fascia. The lateral margin of the fossa is called the falciform ligament. As illustrated in Figure 306, the falciform ligament arises from the pectineus fascia, passes underneath the long saphenous vein, and finally arches medialward over the femoral vein to become attached to the inguinal and lacunar ligaments. That part of the ligament underneath the saphenous is known as the inferior horn and the superior margin as the superior horn.

Descent of Femoral Hernia.—The hernial sac descends through the femoral ring and is directed to the fossa ovalis. The anterior part of the hernia is pressed on and retarded in its downward descent by the falciform ligament. The posterior portion of the sac continues downward and when it reaches the fossa ovalis it is reflected over the falciform ligament and is then directed upward over the inguinal ligament. The points of strangulation of a femoral hernia are at the internal ring in the region of the lacunar ligament, and at the falciform margin.

Anatomical Relationships.—In differentiating inguinal hernia from femoral hernia, the three important landmarks are: the (1) pubic tubercle; (2) femoral artery, and (3) a line drawn from the anterior superior spine to the pubic tubercle corresponding to the inguinal ligament—a hernial mass lying above the inguinal ligament and to the inner side of the pubic tubercle is an inguinal hernia, while a herniating mass inferior to the inguinal ligament and lateral to the pubic tubercle is a femoral hernia.

Normally the obturator artery arises from the hypogastric artery but sometimes from the deep epigastric, in which case it descends behind the pubis to the obturator foramen, passing either medially or laterally to the femoral ring. In a femoral hernia, coexistent with a medially passing obturator artery, the artery is in close relationship to the medial side of the neck of the sac and is liable to injury when the lacunar ligament is incised.

Covering of Femoral Hernia.—The covering of a femoral hernia from within outward are: (1) extraperitoneal fat; (2) femoral septum—consisting of a plug of fat, or lymph node; (3) the femoral sheath (transversalis fascia); (4) the cribiform fascia; (5) superficial fascia; and (6) skin.

Contents of the Sac.—The contents of a femoral hernia is usually omentum. The sac may contain a portion of the bladder with prevesical fat and, at times, small intestine. Rarely, the following viscera have been found: large bowel, appendix, ovary, fallopian tube, kidney, and stomach.

Richter's hernia (partial enterocele) occurs more often in femoral hernia than in any other variety. In this variety only a part of the convex portion of a loop of intestine is caught within the femoral ring (see page 482). In contrast to inguinal hernia, there is a greater incidence of strangulation in femoral hernia due to the rigidness of the femoral ring and the unyielding falciform ligament.

OPERATIVE TECHNIQUE

Subinguinal Approach.—The repair of femoral hernia through a subinguinal approach is simple and easy to perform. The skin incision is either vertical or parallel and below the inguinal ligament. The vertical incision is begun ½ inch above the inguinal ligament and extended downward for a distance of 3 inches. It is about ½ inch lateral to the pubic tubercle so that the incision will cross the center of the fossa ovalis. Before the sac is reached, a variable amount of fat and the long saphenous vein are encountered. Care should be taken not to injure the vein. After the superficial structures have been incised, blunt dissection reveals a fatty

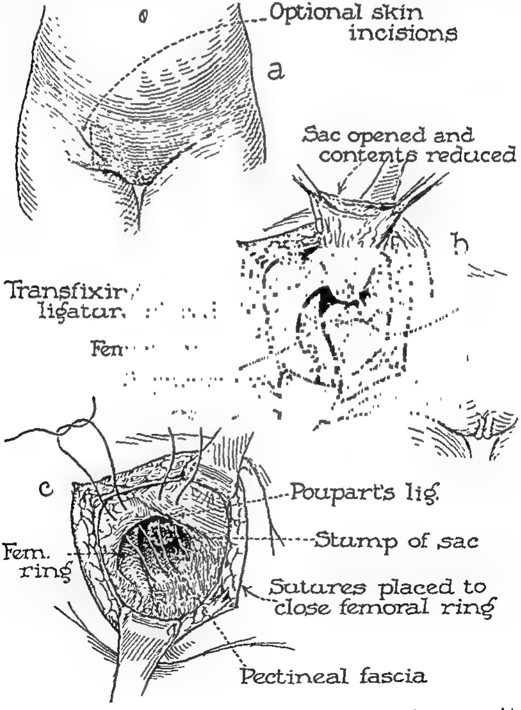


Fig. 308.—Subinguinal approach to a femoral hernia: *a*, skin incision; *b*, isolation and transfixing the sac; *c*, sutures inserted and ready to tie.

tumor. Before proceeding with isolation of the sac, the femoral vessels are identified and retracted out of the field. In large hernia containing a large loop of bowel the falciform ligament may be incised to increase the diameter of the femoral opening.

Where it is possible to do so, the neck of the sac is isolated by blunt dissection. At this stage, if one can be certain of the viability of the contents of the sac, the reduction may be accomplished by placing the patient in the extreme Trendelenburg position. Placing the patient in this position before opening the sac prevents air from being drawn into the abdominal cavity, thus the contents, if not adherent, will be reduced. If there is any question as to viability of the contents, the sac is opened and the bowel is examined well beyond the point of constriction. If the normal color of the bowel returns within a few minutes, it is safe to reduce the bowel. If it does not return to normal, the devitalized bowel is resected and an end to end or a lateral anastomosis is done. In the Richter type of hernia only a small portion of the bowel is necrosed and for this reason local excision of the involved part is sufficient and the lumen is closed in the transverse direction.

In large strangulated hernia which cannot be reduced, the author has found it expedient to divide the shelving edge of the inguinal ligament. This procedure will very often reduce the contents with ease. At times it is necessary to obtain reduction through a low lateral rectus incision. This incision is of special value in that resection and anastomosis, if indicated, can be made much more quickly.

Incarcerated or adherent omentum is a common finding, and reduction in such instances is rather difficult, if not impossible. Reduction may be facilitated by cutting the lacunar ligament, or better the incarcerated omentum may be excised.

After isolation of the sac and reduction of its contents, the neck of the sac is drawn down until the peritoneum beyond the neck comes into view. It is then ligated as high as possible with a transfixing suture and the excess portion of the sac is excised (Fig. 308). Closure at times is more satisfactory with an internal purse-string suture at the neck in addition to the transfixing suture.

Closure of the femoral ring through the subinguinal approach is unsatisfactory, although many methods have been devised. As illustrated in Figure 308, the ring may be closed with mattress sutures approximating the inguinal ligament and the falciform ligament with the pectineal fascia. Bassini accomplishes the same by utilizing three or four interrupted sutures between the inguinal ligament and the pectineal fascia, and two or three sutures to approximate the falciform ligament to the pectineal fascia. Instead of using a mattress suture, a purse-string may be employed. This suture includes the inner portion of the inguinal ligament, the pectineal fascia, and the falciform ligament. This suture is recommended when the hernial sac is small. Regardless of the method used, the surgeon should be careful not to injure the femoral vessels. This can be obviated by retracting the vessels with the index finger while placing the sutures.

The deep layer of the subcutaneous tissue is now approximated with interrupted sutures and the skin is closed in the usual manner.

Inguinal Approach.—Due to the tension on the sutures, and the impossibility of ligating the sac flush with the abdominal peritoneum, the inguinal approach is preferred by many surgeons. Furthermore, the inguinal approach is more anatomical, the sac can be more thoroughly removed, and the femoral ring can be closed with security. In large femoral hernia or in instances when the contents are adherent, a combined inguinal and femoral approach is desirable. In the combined operation the skin incision is made with its first half over the inguinal canal and second half curves downward over the hernia.

The skin incision for femoral hernia through the inguinal route is the same as for inguinal hernia. The fascia of the external oblique is incised in the direction of its fibers (see Chapter on Inguinal Hernia, p. 451), and the conjoined tendon with the spermatic cord or round ligament is retracted. The shelving portion of the inguinal ligament and the transversalis fascia are exposed by blunt dissection. The transversalis fascia is carefully incised parallel with the inferior deep epigastric vessels,

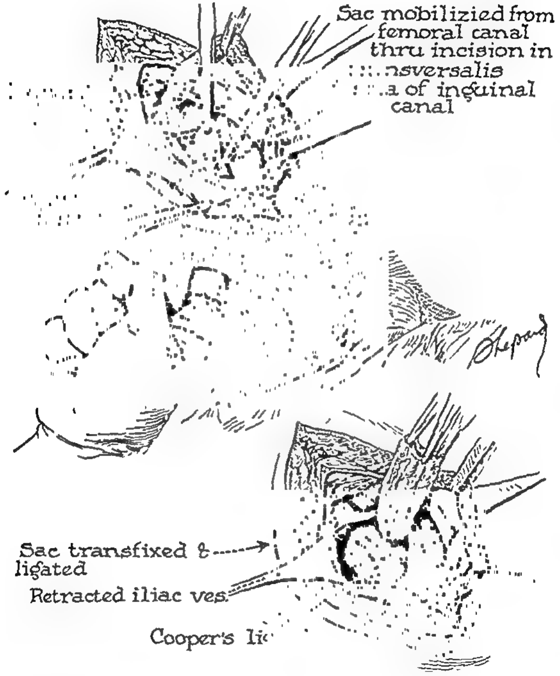


FIG 309.—Inguinal approach for a femoral hernia.

thus exposing the sac proximal to the femoral ring. If the hernia is strangulated an incision is made into the peritoneum just above the neck of the sac. Reduction is then accomplished by gentle traction on the contents of the sac combined with pressure over the mass below the inguinal ligament. If reduction is not possible by this method, the inguinal incision is extended over the hernia and the combined operation is performed.

In non-strangulated hernia, the sac may be delivered after incising and retracting the transversalis fascia (Fig. 309). The sac is opened and contents reduced. The neck of the sac is ligated flush with the peritoneum with or without purse-string suture, and the redundant sac is excised.

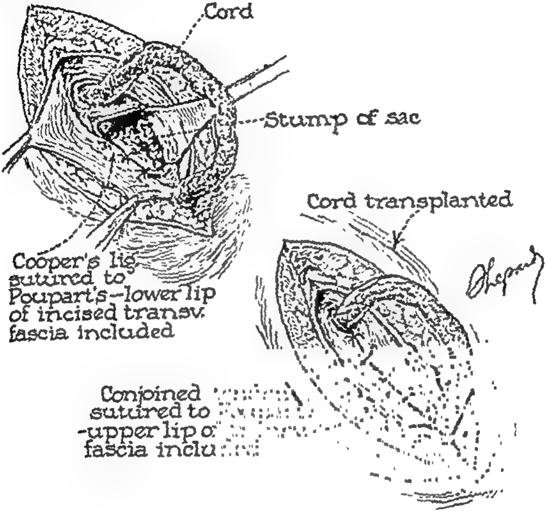


FIG. 310.—Femoral hernia. Method of repair.

The femoral ring is now closed by suturing the shelving portion of the inguinal ligament and transversalis fascia to Cooper's ligament. The latter ligament extends from the pubic tubercle along the pectineal line of the pubis and blends with the periosteum of the bone. To it are attached the pectineus fascia, the lacunar ligament, the transversalis fascia, and Henle's ligament. Three or four interrupted sutures are used for suturing. The stitch in Cooper's ligament should include the periosteum of the bone (Fig. 310). The innermost suture should include the lacunar ligament also. The operation is then completed in a manner similar to an inguinal herniorrhaphy; the cord being transplanted.

QUESTIONNAIRE

1. Give the boundaries of the femoral triangle.
2. What is [illegible]
3. [illegible]
3. [illegible]
4. [illegible] the boundaries of the femoral canal.

5. Why is it wider in the female?
6. What is the fossa ovalis?
7. Give its surgical significance.
8. Describe the descent of a femoral hernia.
9. How would you differentiate an inguinal from a femoral hernia?
10. What are the coverings of a femoral hernia?
11. What are the contents of a femoral hernia?
12. Why is Richter's hernia more frequent in this type of hernia?
13. Why is there a greater incidence of bowel strangulation associated with a femoral hernia?
14. [illegible] oral hernia?
15. [illegible]
16. [illegible]
17. How would you reduce a strangulated hernia?
18. Describe the methods of closing the femoral ring.
19. Give the advantages and disadvantages of the inguinal approach.
20. When is a combined inguinal and subinguinal approach indicated?
21. How would you reduce the sac through the inguinal incision?
22. Describe the closure of the femoral ring through this approach.

CHAPTER 46

ABDOMINAL HERNIA

By A. V. Partipilo

Definition.—Abdominal hernia is a protrusion through the anterior abdominal wall, occurring at points other than the inguinal and femoral openings. From an anatomical point of view, abdominal herniæ occur in the following regions: (1) Umbilical; (2) in the linea alba; and (3) in the linea semilunaris. Incisional herniæ may occur anywhere in the abdominal wall. The term epigastric hernia refers to a protrusion in the linea alba above the umbilicus. Ventral hernia is a protrusion through the anterior abdominal wall, other than the inguinal, femoral, and umbilical openings. Hernia occurring in the linea semilunaris are also known as lateral ventral hernia, or as Spigelian hernia.

UMBILICAL HERNIA

Varieties.—Umbilical herniæ may be classified into three varieties:

1. *Congenital Hernia of the Cord.*—Strictly speaking, this is not a true hernia, because the contents of the sac have never been in the abdominal cavity. The condition should properly be designated as a congenital abdominal eventration, since the viscera have been outside of the abdominal cavity during the greater portion of its embryonal life. The cause of this type of hernia is said to be due to failure of the primitive intestinal loop to withdraw into the abdominal cavity toward the end of the third month of embryonic life.

The diagnosis is usually self-evident; the hernia mass being present as the child is delivered. The hernial sac is thin, translucent, and the size varies from that of an almond to the size of a fetal head. The small intestine is the most usual finding in the sac, however every viscus in the abdomen has been reported as contents.

The choice of treatment depends upon the size of the hernia. Palliative treatment can at times bring about cure of a small congenital umbilical hernia. This consists of application of some type of protective bandage over the hernial sac. However, operative treatment offers the best chance of cure regardless of the size. The radical operation is imperative in the large size hernia, and the operation must be done at once, during the first few hours after birth. If possible the reduction and repair of the hernia should be done without opening the peritoneal sac. The latter is reefed and sewed in place, the muscles and fascia of the abdomen are then sutured to close the defect. If the mass is irreducible, the sac is opened, contents reduced and the excess portion of the sac excised.

2. *Umbilical Hernia in Infants.*—This is also known as infantile umbilical hernia. The condition may be a recurrence of a congential umbilical hernia òr may be due to delayed cicatrization of the umbilicus. The hernia may occur spontaneously as a result of increased intra-abdominal pressure from any cause. The size of the hernia varies from that of the tip of the finger to a very large mass.

3. *Umbilical Hernia in Adults.*—This type of hernia is usually due to a gradual yielding of the cicatricial tissue closing the ring. Obesity and pregnancy being the principal causes.

Surgical Anatomy.—The umbilical ring closes soon after birth; the umbilical arteries and veins are converted into connective tissue. The ring contracts and a firm scar results with fibers running across the median line and others interlacing

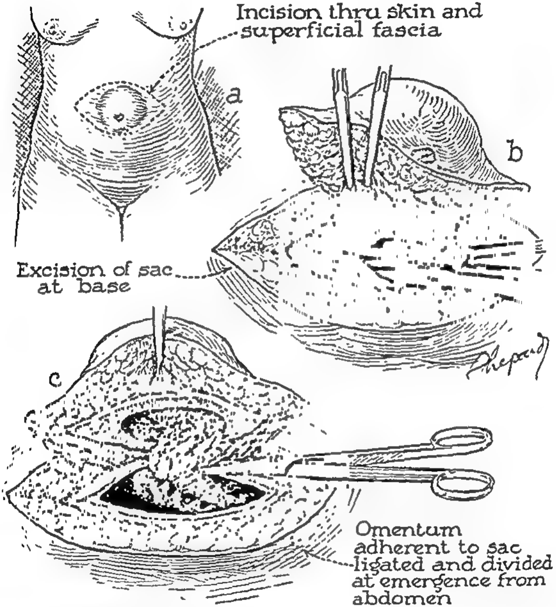


Fig. 311.—Umbilical hernia: (*a*) the incision; (*b*) the sac is opened near the neck; (*c*) separating omental adhesions.

the lateral aspects of the umbilicus. The weakest portion of the ring is its upper margin, the lower margin being well protected by the two obliterated umbilical arteries. The hernia starts as an outpouching at the upper angle at the site of the obliterated omphalo-mesenteric artery. The direction of force in this area is outward and downward and as the sac increases in size, the lateral edges of the recti muscles are pushed aside. The umbilical sac is most often lateral or inferior to the bulk of the hernia.

Operative Technique.—Strangulation in umbilical hernia is a common finding, and this modifies the treatment to a certain extent. In non-strangulated hernia, the Mayo operation is the procedure of choice. It consists of a transverse, elliptical incision (Fig. 311, *a*) which is carried down to the aponeurosis of the recti muscles. The superficial fatty tissue is dissected free from the aponeurosis for a considerable distance on all sides of the hernial ring. It is important to expose the anterior sheaths of the recti muscles, else the repair may be imperfect. The sac is now opened near the ring border and a finger is inserted into the sac to determine whether

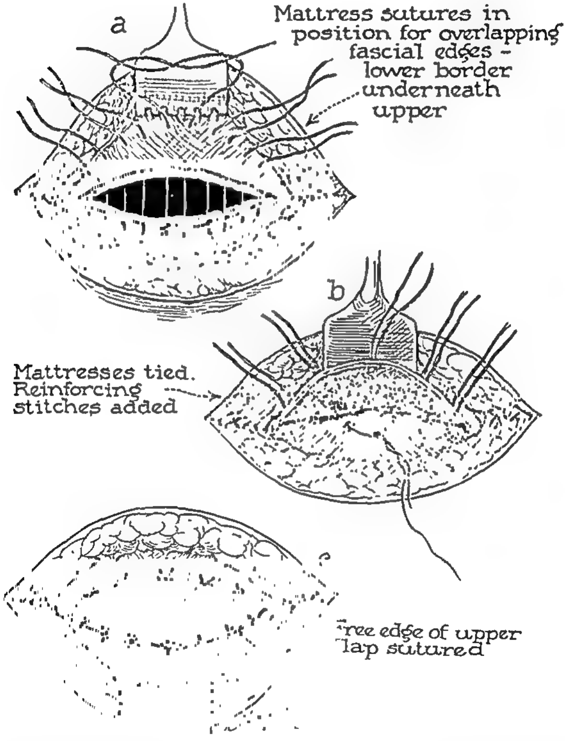


FIG. 312.—Transverse overlapping of the fascia (Mayo method): (*b*) sutures drawn tight and tied, thus pulling the lower flap behind the upper flap; (*c*) the free edge of the upper flap is sutured to the lower flap.

omentum or viscera are adherent. Adherent omentum is best removed by ligation and excision at the ring level (Fig. 311, c). With a finger as a guide (a spatula may be used) the sac is excised together with the overlying skin. A collar of the sac about ½ inch, or more, should be left to permit easy closing.

The ring is now enlarged by transverse incisions of the rectus fascia and peritoneum and closed by imbrication as advocated by Mayo. The fascia and peritoneum is overlapped by means of interrupted sutures, so that the lower layer lies underneath the upper layer. The first sutures are mattress, one beginning approximately ¾ inche from the free edge of the upper flap from without in then through the edge of the lower flap in the same manner, being brought back through the lower flap from within out and so through the upper flap (Fig. 312, a). After placing an adequate number of these sutures they are tied, beginning with the center one. As illustrated in Figure 312, b, the line of this first series of sutures is reinforced by interrupted sutures approximating the flaps between the mattresses. The free edge of the upper flap is then sutured with interrupted sutures (Fig. 312, c).

In strangulated hernia the relief of the obstruction is the primary consideration of the surgeon. In gangrenous obstruction the involved segment is resected and an end to end or a lateral anastomosis is done. The repair of the hernial defect is of secondary consideration. If the hernial sac is small, and the condition of the patient is favorable, herniorrhaphy may be done. However, if the obstruction is of long duration the relief of the obstruction by incising of the ring at the neck of the sac is the procedure which offers the least risk. Secondary repair of the defect may be done at a later date when the condition of the patient allows it. Furthermore, gangrenous obstruction of long duration may require excision of the bowel and a double barrel enterostomy through the hernial opening. Herniotomy is also indicated in the obvious bad risk patients without gangrenous or strangulated bowel. These are the old, debilitated and obese patients with large, often strangulated, herniæ. In these cases the constriction is relieved by incising the ring of the sac, thus converting the sac and the peritoneal cavity into one.

Ventral Hernia.—A ventral hernia is a protrusion through the anterior abdominal wall other than the inguinal, femoral, and umbilical openings. When the hernia occurs between the umbilicus and the ensiform, it is known as epigastric hernia. Epigastric hernia may consist of a small mass of subperitoneal fat, without any sac; the subperitioneal fat may have the peritoneum attached to it, or a true hernia with a sac may be found a short distance of each other and are separated only by a narrow band of fascia. They are rarely of large size and for this reason present diagnostic problems. A lipoma in the linea alba may have all the characteristics of an omental hernia, even the impulse upon coughing; a lipoma is more movable than a true hernia, and if the tumor is superficial to the fascia the impulse is not prominent or is absent. In hernia the connection with the deeper layers of the abdominal wall or the ring can usually be demonstrated.

Hernia in the linea semilunaris (Spigel's hernia) above the semilunar fold of Douglas are extremely rare. The great majority of them occurring at the level of the folds of Douglas and below the deep inferior epigastric artery. Those appearing below the deep epigastric artery and the lateral border of the rectus are considered as direct inguinal herniæ. In 1942 River found only 112 reported cases of true spontaneous Spigelian herniæ to which he added four cases of his own. A hernia in the linea semilunaris is usually small, rarely attaining the size of an orange. The hernial opening is most frequently found at the junction of the spino-umbilical

line and the linea semilunaris, where the posterior rectus sheath forms the fold of Douglas. This is the weak point of the semilunar line, hence the frequency of herniæ in this area. The sac is mushroom-shaped and may contain omentum, small intestine, or large intestine. The hernia is very often interstitial; beneath the external oblique fascia and in front of the transversalis fascia. This type is termed "Masked Hernia" by Macready. In some cases the diagnosis may be obvious due to the presence of a protruding mass or very difficult when the hernia is of the interstitial type.

Of the abdominal herniæ, the incisional or postoperative is the most prevalent type. Postoperative herniæ usually occur most commonly after longitudinal incisions, especially when the nerve supply to the rectus muscle has been destroyed. As a result of better understanding of the anatomy of the abdominal wall and the nutritional requirements of the patient, incisional herniæ are less common at the present time. The surgeon is referred to Chapter 43 for the rules in making an abdominal incision, if he wishes to avoid some of the common causes of postoperative hernia. Also, he should review the subject of wound healing (Chapter 1), and develop a good suture technique (Chapter 43).

Operative Technique.—The surgical anatomy of the anterior abdominal wall with which the surgeon is concerned in treating abdominal hernia, has been discussed in a previous chapter. The site and size of the hernia modify to a certain extent the technique of repair of these herniæ. The following methods are applicable for the various types of abdominal herniæ.

1. The Mayo technique for the repair of umbilical hernia is well adapted for epigastric hernia. It is also recommended for small incisional hernia.

2. In small incisional herniæ, such as those which occur after draining muscle splitting incisions, repair may be done by dissecting the component parts of the abdominal wall and closed in layers with or without overlapping.

3. In large spontaneous or incisional hernia an elliptical incision, including the scar if present, is made about the mass and the incision is carried down to the fascia. The latter is exposed well on all sides by sharp dissection. The sac is incised near the edge of the ring, everted, and any adherent contents freed. The excess portion of the sac is excised and the layers of the abdominal wall on either side are separated, and the closure is made as in umbilical hernia. If the hernial ring is too large, making it impossible to do the Mayo operation, the ring may be closed by a combination of a vertical and transverse closure of the fascia.

4. In the lower part of the abdomen, the hernial ring is oval in the vertical direction, hence the incision and repair should be done in the vertical direction. The fascia is repaired by imbrication, if possible.

5. Many ingenious methods have been devised for the repair of large abdominal defects. This includes the use of heterogenous and homogeneous grafts. All these cases present individual surgical problems which may tax the ingenuity of the master surgeon. No one particular method will satisfy the needs of a specific case, hence the surgeon must be well informed and apply his ingenuity to fit the exigencies presented.

QUESTIONNAIRE

1. Define: abdominal hernia.
2. Give the three varieties of umbilical herniæ.
3. Discuss congenital hernia of the cord.

4. Is it a true hernia? Why?
5. Give the surgical anatomy of the umbilicus.
6. Describe the Mayo technique for the repair of umbilical hernia.
7. Define: ventral hernia.
8. Give the characteristic features of an epigastric hernia.
9. What is "Spigel's Hernia"?
10. Discuss the surgical treatment of ventral herniæ.

REFERENCE

1. River, Louis P.: Spigelian Hernia, Ann. Surg., Sept., 1942, 116, 405-411

CHAPTER 47

HYDROCELE

By A. V. Partipilo

GENERAL CONSIDERATIONS

Hydrocele may be defined as a collection of fluid in some part of the processus vaginalis. The composition of the fluid is inflammatory in nature. Hydrocele may occur as an acute or chronic condition following inflammation of the testicle or epididymis, especially the latter. Acute hydrocele is most often associated with gonorrhea and tuberculosis, both of which involve the epididymis rather than the testicle. It may also occur in the course of acute infectious diseases, such as typhoid

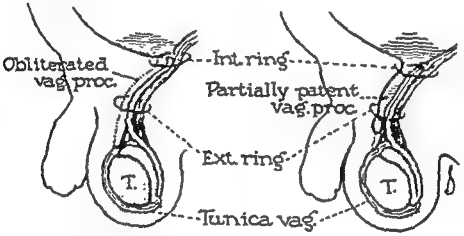
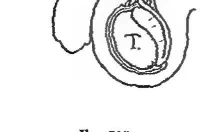


Fig. 313 Fig. 314

Fig. 313.—Normal occlusion of the vaginal process.
Fig. 314.—Partially patent process with normal tunica vaginalis.



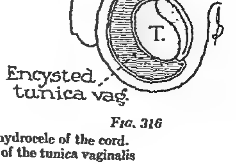


Fig. 315 Fig. 316

Fig. 315.—Encysted hydrocele of the cord.
Fig. 316.—Hydrocele of the tunica vaginalis

32

fever, mumps, etc. In the uncomplicated case the hydrocele subsides within a period of a few days to two weeks. Chronic hydrocele may be a manifestation of the after effect of the acute form, however, no satisfactory explanation has been given for it.

Hydrocele in young people is due to accumulation of fluid in the unobliterated processus funicularis and is therefore of the congenital variety. There are five possible anatomical varieties of hydrocele. These are:

1. *Funicular Type.*—This consists of a partially patent funicular process with a normal vaginalis (Fig. 314). This variety communicates with the peritoneal cavity, therefore, a passage is present through which abdominal content may pass into the sac. This type is more properly considered as a hernia since the wide communication with the peritoneal cavity does not permit accumulation of fluid (Fig. 320). If the communication permits the intestine to pass into the sac, the

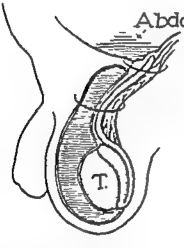


FIG. 317

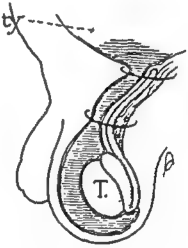


FIG. 318

FIG. 317.—Complete hydrocele of the cord and testis.

FIG. 318.—Patent vaginal process. The tunica vaginalis in this instance communicates with the peritoneal cavity.

hydrocele may be of the intermittent type. In this instance the fluid may escape into the peritoneal cavity at intervals such as when the patient is lying down or by pressure. It is very often confused with the funicular type associated with hernia. The latter is distinguished by the greater ease in its reduction.

2. *Hydrocele of the Cord.*—This is also known as an encysted hydrocele. In this type the funicular process fails to become obliterated between the testicle and the peritoneal cavity (Fig. 315). This variety is very often mistaken for an encysted hydrocele of the epididymis which is a dilatation of the spermatic ducts. Boyd has termed this condition a spermatocele. It forms a small globular swelling at the upper end of the epididymis. The cyst is usually single, but may be multiple or multilocular. Hydrocele of the cord is commonly found in infants and is very frequently mistaken for hernia. The presence of an irreducible fluctuant mass distinguishes a hydrocele from a hernia.

3. *Hydrocele of the Tunica Vaginalis.*—This type is more often secondary to diseases of the testis or epididymis (Fig. 316).

4. *Complete Hydrocele of the Funicular and Vaginal Process.*—The process vaginalis is occluded at the internal ring only (Fig. 317). Not infrequently a hernia

is associated with this type of hydrocele. The infantile or encysted variety was described by Hey, who called it "infantile," probably because the case he observed occurred in an infant. Later, Copper found this variety of hernia associated with a hydrocele and designated it as an encysted hernia. The latter term is more appropriate because it is anatomical. In the encysted hernia the funicular process is closed at the internal ring, but remains open below. The hernia passes down behind the opened process (Fig. 321), or the hernial sac may invaginate into the sac of the hydrocele (Fig. 322). In either case a form of hydrocele is associated with the hernia.

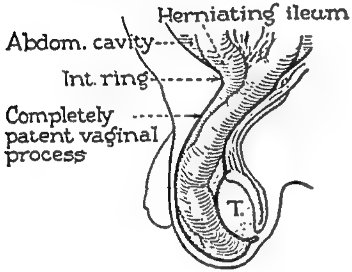


FIG. 319.—Complete congenital hernia.

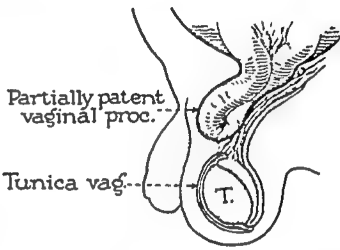


FIG. 320.—Incomplete funicular hernia.

5. *Patent Vaginal Hydrocele.*—The tunica vaginalis in this type communicates with the peritoneal cavity (Fig. 318). This is also of the intermittent variety. If the communication between the sac and peritoneum is small, it permits fluid to escape into the peritoneal cavity upon pressure or upon standing. With a larger peritoneal opening, it becomes a complete congenital hernia (Fig. 319).

Treatment of Hydrocele.—Treatment of a hydrocele depends upon the conditions present. The treatment of the acute and symptomatic variety consists of treating the underlying pathology. These usually subside with rest, immobilization, and specific therapy if indicated. Similarly, a hydrocele in a child rarely requires radical treatment. If the hydrocele communicates with the peritoneal cavity it should be treated as a hernia.

Treating the hydrocele by aspiration and injection of a sclerosing solution has its advocates. In the past, a great number of substances have been used. Among these, iodine, phenol, sodium chloride, quinine hydrochloride, sodium morrhuate, quinine dihydrochloride and a host of others have been used with varying results. Kilbourne and Murray concluded that a sclerosing solution must possess the following properties:

1. It must be painless.
2. It must not cause disability.
3. It must be efficient in destroying all hydrocele without recurrence.
4. It must not be dangerously toxic.
5. It should be bactericidal.

6. It must not subject the patient to the danger of hemorrhage into the sac following the injection.

Kilbourne and Murray advocated a solution of quinine hydrochloride, urethane and diothane. Injection treatment may be employed with safety in simple asymptomatic variety of hydrocele. The procedure is simple and may be carried out in the office. In certain cases when injection or operative treatment is for some reason contraindicated, simple tapping alone may be done as a palliative measure. Injection treatment is contraindicated in the acute hydrocele. It is also contraindicated when the sac communicates with the peritoneal cavity or where there is pathology present in the testicle or the epididymis.

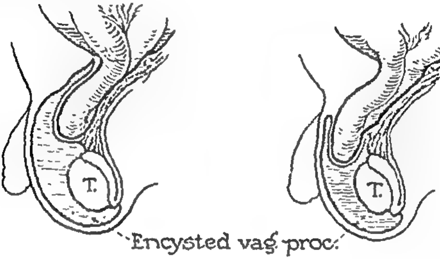


Fig. 321 Fig. 322

Fig. 321.—Infantile hernia; the sac passing behind the tunica.

Fig. 322.—Infantile hernia; the sac invaginating the tunica

Operative Treatment.—*There are numerous procedures for the radical cure of a* hydrocele. The common objective of all the methods is either to extirpate the sac entirely, evert the sac, or perform a partial excision of the sac with eversion. Regardless of the method used, hemostasis must be absolute. Every vessel must be carefully tied if secondary hemorrhage is to be avoided. Secondary hemorrhage is not only troublesome, but infection may superimpose upon it, and in a few instances loss of the testicle has resulted.

For a hydrocele with a thin-walled sac the Bottle operation is the one of choice. If the sac is large, an adequate portion of it is excised to permit approximation of its edges behind the epididymis without leaving too much redundancy. In this operation it is important to make sure that the sac is everted from pole to pole otherwise a pocket will remain which will refill after the operation. When the sac wall is unusually large and thick, partial or complete excision without eversion gives the best results. If the hydrocele is associated with a hernia the operative procedure consists of excision of the hydrocele sac and a herniorrhaphy. Hydrocele communicating with the peritoneal sac is treated as a complete hernia and the hydrocele sac is excised.

Technique of the Bottle Operation.—After the operative field has been properly prepared and cleansed, an incision is made through the anterior surface of the scrotum (Fig. 323, *a*). The length and location of the incision will depend upon the

size and extent of the hydrocele. For a hydrocele of the tunica vaginalis a scrotal incision about 2 inches long is sufficient. If the hydrocele extends into the inguinal canal, the scrotal incision is extended over the inguinal region. Encysted hydrocele of the spermatic cord and hydrocele associated with complete hernia are exposed by inguinal incisions as for inguinal hernia.

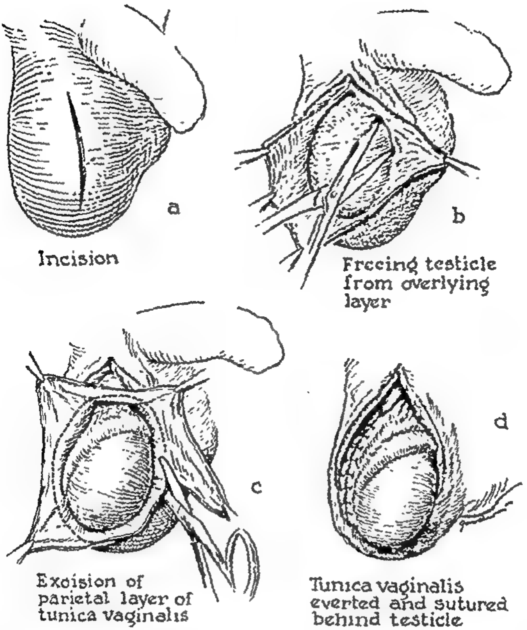


FIG. 323.—Operative treatment of hydrocele. (a) the skin incision; (b) method of freeing the adherent sac; (c) excision of excess portion of the sac; (d) tunica everted and sutured behind the testicle

The skin incision is carried down through all the layers of the scrotum. Before proceeding, clamp and ligate all bleeding points. This is a vascular operation and every attempt should be made to control hemorrhage with every step. The sac is now bluntly dissected free from pole to pole and delivered into the wound. In the original "Bottle Operation," described by E. Wyllys Andrews, the sac is treated as follows:

An incision is begun at the extreme upper pole of the sac. This is made vertically

6. It must not subject the patient to the danger of hemorrhage into the sac following the injection.

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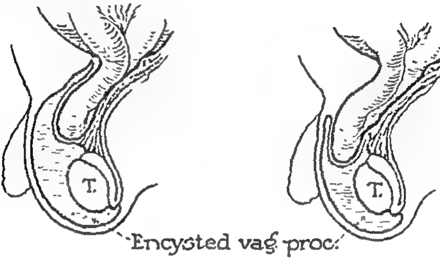

Fig. 321 Fig. 322

Fig. 321.—Infantile hernia; the sac passing behind the tunica.

Fig. 322.—Infantile hernia; the sac invaginating the tunica.

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For a hydrocele with a thin-walled sac the Bottle operation is the one of choice. If the sac is large, an adequate portion of it is excised to permit approximation of its edges behind the epididymis without leaving too much redundancy. In this operation it is important to make sure that the sac is everted from pole to pole otherwise a pocket will remain which will refill after the operation. When the sac wall is unusually large and thick, partial or complete excision without eversion gives the best results. If the hydrocele is associated with a hernia the operative procedure consists of excision of the hydrocele sac and a herniorrhaphy. Hydrocele communicating with the peritoneal sac is treated as a complete hernia and the hydrocele sac is excised.

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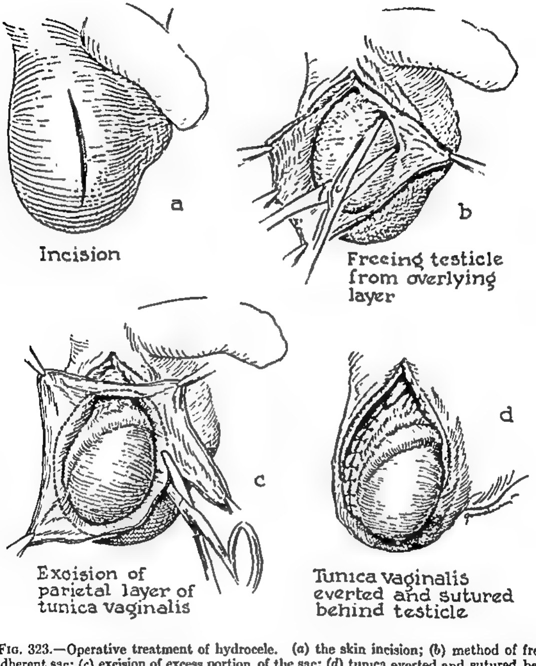

Fig. 323.—Operative treatment of hydrocele. (*a*) the skin incision; (*b*) method of freeing the adherent sac; (*c*) excision of excess portion of the sac; (*d*) tunica everted and sutured behind the testicle

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An incision is begun at the extreme upper pole of the sac. This is made vertically

on the anterior border and about 1 inch (2 cm.) long. It is enlarged by stretching to the extreme end along the cord if the first incision did not do this. When the sac is emptied, it is like a bottle with a small hole at the top; hence the term "Bottle Operation." Andrews described the method of emptying the sac as follows: "Dilating this slightly with one or two fingers, the orifice is held open and the testis is *pushed up into it with the other hand or the two thumbs. In a moment it can be squeezed through, and the whole sac will instantly be everted with the small button*-hole so closely surrounding the cord that it is scarcely visible." . . . "The short incision contracts, so as to fit around the cord, and the whole sac by its elasticity seems to collapse around the epididymis with its white serous surface almost as smooth as that of the testis." The everted sac remains everted without use of stitches. This method is applicable for the thin-walled hydrocele.

In the Jaboulay's operation the sac is opened and the contents emptied. After ligating bleeding points, the tunica is everted and its edges are sutured behind the testicle (Fig. 323, *d*). Thus the process vaginalis is turned inside out and exposes its entire secreting surface to the wound which will usually absorb the secretions from the sac. Eventually the secreting surface is destroyed. If the sac is too large, the excess portion may be excised (Fig. 323, *c*). At times the sac may be adherent to the overlying testicle, and the sac must be freed from it. This should be done with care to avoid injury to the testis (Fig. 323, *b*).

The operation is completed with or without drainage. If the sac is large, a cigarette drain is placed into the wound for twenty-four hours. This should be brought at the upper end of the incision or through a counter incision. The dartos muscle is closed with interrupted sutures and the skin is closed with a continuous silk or dermal suture. The scrotum should be kept elevated on a pillow for forty-eight hours postoperatively.

QUESTIONNAIRE

1. Define: hydrocele.
2. Discuss the etiology of hydrocele.
3. Describe the following varieties: (*a*) Funicular type. (*b*) Hydrocele of the cord. (*c*) *Hydrocele of the tunica vaginalis.* (*d*) *Complete hydrocele of the funicular* and vaginal process. (*e*) Patent vaginal hydrocele.
4. What is an encysted hydrocele?
5. What is a spermatocele?
6. Define: infantile hydrocele
7. Discuss the injection treatment of a hydrocele.
8. What is the objective of the operative treatment?
9. When is the "Bottle operation" indicated?
10. Describe the technique of the Bottle operation.
11. How did the term "Bottle operation" originate?
12. What is the principle of Jaboulay's method?
13. How would you treat a hydrocele with a large sac?

CHAPTER 48

VARICOCELE

By A. V. Partipilo

GENERAL CONSIDERATIONS

Varicocele may be defined as a varicosity of the spermatic vessels, forming a swelling in the scrotum that feels like a bag of worms, and is accompanied by a dragging sensation.

Two types of varicocele are recognized, the symptomatic and idiopathic. The former is rare and usually occurs in older people. It develops suddenly and may attain a large size. The symptomatic variety is usually secondary to obstruction of the spermatic vein as a result of pressure exerted on the vein either within the inguinal canal or the abdominal cavity. The condition is more often due to tumors of the kidney; however, it may result from any peritoneal tumor, a hernia, by wearing a truss or from a tumor in the inguinal canal. A diagnostic sign of obstructive varicocele which distinguishes it from the idiopathic type is the slow emptying of the spermatic veins when the patient is placed in the prone position. The treatment of the symptomatic varicocele consists of treating the underlying causative factor.

The idiopathic varicocele is the common type. It occurs in young individuals during their greatest sexual potentiality. It develops slowly, affects the left side most often and is of unknown etiology. The left sided preponderance has been explained on the fact that the left vein empties at right angles into the renal vein, whereas the right empties obliquely into the vena cava. The right vein possesses efficient valves while the left is devoid of valves at its opening into the renal vein. The left vein may also be compressed by the sigmoid since it lies underneath this portion of the colon. It has also been explained on the fact that the left testicle hangs lower than the right, hence a longer spermatic vein.

The diagnosis of idiopathic varicocele is made on the findings upon inspection of a low hanging left testicle with a dilated and relaxed scrotum; palpation of a "bag of worms" and rapid emptying of the veins in the prone position in young individuals.

Treatment.—Therapeutically, idiopathic varicocele may be divided into three groups: (1) the asymptomatic in which operation is not indicated; (2) scrotal enlargement with symptoms of dragging sensation. If conservative treatment with a suspensory fails to bring relief, operation is indicated; (3) the third group consists of those with sexual neurosis with or without symptomatic varicocele. Operation is not advisable in this group of cases.

Operative Technique.—A skin incision about 1½ inches long is made along the course of the spermatic cord with the external abdominal ring at its center (Fig. 324, *a*). The incision is carried down to the spermatic cord. The external abdominal ring is identified with the cord emerging through it. The cord is now carefully lifted from its bed and the operator identifies by palpation the various struc-

tures of the cord. The *vas deferens* lying behind the veins and artery is distinguished by its hard, cord-like consistency when the cord is held between the index finger and thumb. The next important structure is the spermatic artery which should be identified, if possible. It should never be divided unnecessarily. Cases have been reported of testicular atrophy as a result of severing the artery. If the spermatic artery cannot be identified it is much safer to remove fewer veins.

After identifying the vas and artery, make a longitudinal incision along the coverings of the cord to expose the spermatic veins (Fig. 324, *c*). Three groups of veins are found in this area. These are: (1) an anterior group where the spermatic

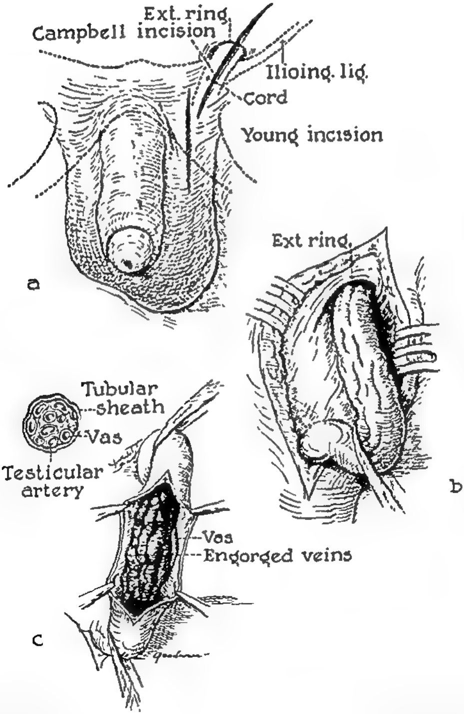

FIG. 324.—Operation for varicocele. (*a*) the skin incision; note that it bisects the external abdominal ring; (*b*) the cord is freed from its bed; (*c*) an incision is made through the various coverings of the cord.

artery may be found; (2) a middle group which has the vas deferens; and (3) a posterior group. Any or all of these may be dilated, however the anterior spermatic group is the most frequently affected. If all the veins are affected the anterior group is usually most extensively involved.

The anterior and middle groups of veins are now isolated from the vas and artery for a distance of about 3 inches. Now apply clamps at the distal and proximal extremities of the isolated veins, ligate below the clamps and divide the veins

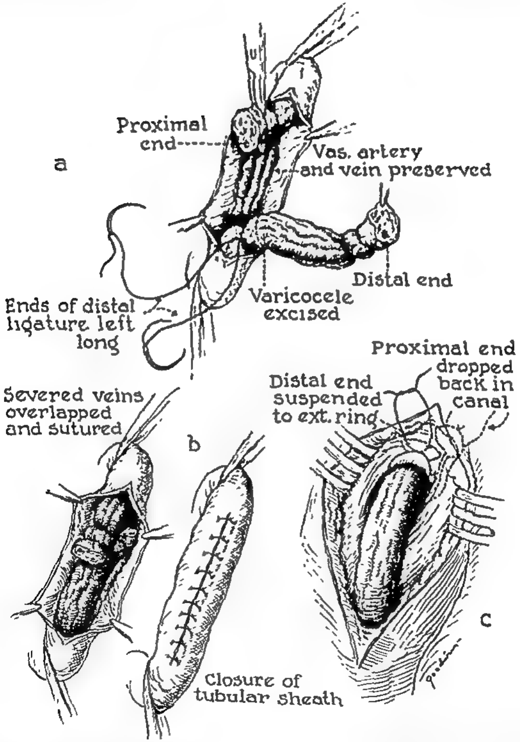

FIG. 325.—Method of suspending the veins. (*a*) ligation and excision of the veins; (*b*) the two ends are simply tied together; (*c*) suspension of the distal end by suturing it to the external abdominal ring. Figure between *b* and *c* shows the cord coverings sutured with interrupted stitches

between the ligatures and the clamps (Fig. 325, a). To obviate the possibility of the ligature to slip, it is preferable to double ligate each end. The divided ends can now be treated in various ways. The two ends may be tied together as illutrated in Figure 325, *b*. This is Young's method. Another method consists of dropping the proximal stump back into the inguinal canal and suspending the distal one to the external abdominal ring (Fig. 325, *c*). Both of these procedures are feasible only when the pampiniform plexus of veins has not been entirely excised. If the plexus has been totally removed, the procedure suggested by Londres may be used. This consists of securing a flap of external oblique fascia, ¾ inch wide and 2 inches long with its pubic insertion left intact and the loose end sutured to the tunica vaginalis of the testis. In all of these methods the object is to suspend the testicle. If the scrotum is extremely large and pendulous, the redundant portion may be removed.

Before the wound is closed, complete hemostasis must be assured. The most frequent complication following this operation is hemorrhage; therefore the importance of hemostasis cannot be stressed too vigorously. The cord covering is closed with a continuous or interrupted suture and a dermal or silk suture is applied to unite the skin edges. Drainage is not necessary. A firm scrotal bandage is applied and a suspensory should be worn for two or more weeks after the patient *leaves the hospital.*

QUESTIONNAIRE

1. Define: varicocele.
2. What is the most common cause of the symptomatic variety?
3. Discuss the anatomical factors which may cause the idiopathic type.
4. What is the diagnostic sign which distinguishes the obstructive variety from the *idiopathic?*
5. Discuss the operative indications for removal of varicocele.
6. Describe the technique for the operative treatment of varicocele.
7. What important structures are to be avoided?
8. How many groups of veins are there, and what are their relations to the vas deferens and the spermatic artery?
9. What is the primary objective of excising a good portion of the veins?

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CHAPTER 49

GASTRIC SURGERY

By A. V. Partipilo

GENERAL PRINCIPLES

Gastric Physiology.—The stomach is the largest organ of the digestive system, measuring when fully distended from 10 to 11 inches in length and about 4 inches in diameter. This large size is a natural necessity, since the stomach acts as a storehouse during the process of macerating, churning, and preparing the food for its descent, and further digestion in the intestinal canal. Because of the nature of this function, the stomach bears the brunt of attack from raw, undigested, and at times injurious food. It can be considered as the laborer of the digestive system, and in common with workers, it is subjected to traumatic injuries. Furthermore, it is subject to psychic control which, if abnormal, produces physiological and motor disturbances that may undermine and interfere with normal gastric function. It is also affected by and responds to any abnormal condition affecting the rest of the digestive and biliary systems.

Gastric Secretion.—The stomach does not assimilate food to any great extent, however, it is a great secretory organ, capable under abnormal conditions of secreting as much as 5 liters of fluid during a twenty-four-hour period. It has been estimated that there are about 35,000,000 secretory glands in the stomach. These are found throughout the entire extent of the mucosa, and are composed of specialized cells which differ in structure and in the nature of their secretions in various portions of the stomach.

The glands are arranged as tubules which consist of a body which reaches nearly to the muscularis mucosæ and a short superficial neck. Each tubule opens into a little pit, the gastric foveola, through which the juice is discharged. The tubules are lined by three types of cells:

a. Chief cells of the neck, or mucus cells, which produce an alkaline fluid rich in mucin. These cells are found chiefly in the pyloric and cardiac portions of the stomach, and less numerous in the fundus and body. Gastric mucin has a pH of 7.0 to 7.5 which serves to neutralize gastric acidity because of its high combining power. It also is said to have anti-peptic properties due to its mucotin-sulfuric acid.

b. The chief cells of the body of the tubule contain zymogen granules which are the precursor to pepsin. They are found mostly in the fundus of the stomach. Pepsin initiates the splitting of the meta-proteins into proteoses and peptones. Pepsin is active in an acid medium only; its optimal pH is 1.7, however the exact optimum activity differs with the nature of the protein being digested. Two other enzymes, rennin and lipase, are also assumed to be secreted by the chief cells of the fundus of the stomach. Rennin is a milk curdling enzyme which is abundant in infants, but low or absent in the adult. It acts on casein with the formation of paracasein and whey albumose. They are soluble products which in combination

with calcium forms calcium paracasein. The latter is thrown out of solution to form the curd. According to Alvarez, the reason for milk-curdling is so that the milk can be kept in the stomach long enough for it to undergo peptic digestion. If milk were to remain in its liquid state, it would promptly leave the stomach. Gastric lipase is a weak fat-splitting ferment or enzyme. It differs from the lipase of the pancreas in being active in an acid medium, its optimum pH varying from 4 to 5. Because of its weak action and due to the fact that a rising acidity renders it inactive, gastric lipase is of little practical importance.

c. The third type are the parietal cells which are found lining the entire extent of the tubule, lying between the chief cells and the lining membrane. They produce hydrochloric acid and water of the gastric juice and for this reason they are known as oxyntic cells and acid cells. Within the cells there is a system of canals through which the acid secretion passes between the chief cells to the tubule lumen. The parietal cells are found exclusively in the fundus and body of the stomach. None are found in the pyloric and cardiac portions. In these regions the tubules are lined by mucus cells which produce a slightly alkaline secretion, rich in mucus. It is now widely believed that the acidity of the gastric juice as it leaves the glands remains constant, regardless of the rate or volume of the secretion. According to Pavlow, the purest juice obtained with the help of histamine has a concentration of about 0.6 per cent hydrochloric acid, or a pH of about 0.9. Values less than this are due to mixtures of dilution and neutralization by the alkaline juices.

Composition of Gastric Juice.—Gastric juice obtained through a fistula has the following composition (modified after Carlson):

Acidity: Free HCl, 0.40 to 0.50 per cent.
Total acidity, 0.45 to 0.60 per cent.
pH 0.9 to 1.5.

Gastric juice obtained by the stomach tube during the interdigestive phase shows a free HCl of 0 to 0.1095 per cent, or from 0 to 30 degrees. (Degree of hydrochloric acid is a term expressing the number of cubic centimeters of decinormal sodium hydroxide solution required to neutralize 100 cc. of secretion). The total acidity varies from 10 to 50 degrees, or from 0.036 to 0.182 per cent. Total acidity includes free hydrochloric acid, hydrochloric acid combined with protein and organic salts.

Stomach contents after the usual test meal has a variable acidity, depending upon the age and sex of the patient, and gastric disturbances. Vanzant observed that it increases rapidly from childhood up to the age of twenty years when adult values are obtained. Free HCl varies from 45 to 65 degrees, and the total from 50 to 100 degrees. The gastric juice secreted in an empty stomach after injection of histamine has a total acidity of from 80 to 140 degrees. At the height of digestion of a mixed meal the acidity approaches that which occurs after histamine.

Solids: Organic, including mucin and the various ferments, 0.42 to 0.46 per cent.
Inorganic, 0.13 to 0.14 per cent.
Specific gravity, 1.006 to 1.009.
Total nigrogen, 0.051 to 0.075 per cent.

Mechanism of Gastric Secretion.—Physiologists have divided the various phases of gastric secretion into two periods: the first, the period of interdigestive or continuous secretion, the second, the period of digestive secretion.

The First Period.—This is the period when secretion occurs during the interdigestive phase when there is no food in the stomach. Pavlov believed that the secretion of gastric juice was intermittent and that the resting stomach did not secrete and if acid was present it was due to psychic factors. Others have shown that variable amount of secretion takes place during this period. According to Bloomfield, Chen, and French, patients with duodenal ulcer manifest a "hypernormal continuous secretion"; *i.e.*, their stomachs continue to secrete copious amounts of acid juice after they have been emptied. This secretion is not entirely abolished by relatively large doses of atropine as is the normal continuous secretion. Ivy suggests that in patients with peptic ulcer some abnormal process is concerned, or that some process which plays a minor role in the normal state has been activated. It is possible that histamine is produced excessively by an irritated mucosa, since the secretory response to the ordinary dose of histamine used is not prevented by ordinary doses of atropine (Ivy and Bachrach). The amount of secretion varies. The nature of the stimuli producing the continuous secretion is unknown.

The Second Period.—This is the period of gastric secretion and is divided into three phases: first, the cephalic phase; second, the gastric or chemical phase; and third, intestinal phase.

First, The Cephalic Phase: This is also known as the psychic phase. The secretion in this phase is due to a stimulus derived from sight, smell, taste, and thought of food. Pavlov was the first to demonstrate the psychic secretion of gastric juice in dogs. This was done through a pouch of the stomach which was free from the main cavity of the stomach. Through the fistula in the pouch pure gastric juice uncontaminated with food was collected. The amount of juice secreted during the cephalic phase varies considerably in the same subject. According to Ivy the amount is from 50 to 150 cc. within twenty minutes, in man. Through "sham feeding," when the food after being swallowed simply comes out from an esophageal opening in the neck, a profuse amount of gastric juice is secreted within a few minutes. The secretion is rich in pepsin. It was demonstrated that the presence of food in the stomach is not necessary to produce the psychic phase, nor is it essential that the food enter the mouth to produce the response; appetizing food which can be seen or smelled is sufficient.

Division of the vagus nerves completely will annul the cephalic phase and thereby reduce the total secretion of gastric juice induced by a meal. It is also prevented by atropine. The quantity of juice varies with different foods, however the most important factor is its palatability. Meat produces the greatest amount of juice rich in pepsin; bread and other unsavory foods produce less juice and less ferments. Alvarez points out that the cocktail, the soup, and the *hors d'oeuvres* with which so many people begin their dinner, and the meat extracts, sauces, and condiments which they add to their food are, for the most part, strong stimulants to secretion, and capable, perhaps, of substituting for a deficient or absent psychic juice.

Second, The Gastric or Chemical Phase: In this phase the secretion occurs as result of direct stimulation of the gastric mucosa by food present in the stomach. When food is present in the stomach the secretion continues for a much longer time than is accounted for by the cephalic phase. Two factors are responsible for this phase.

1. Mechanical Stimuli.—The flow of gastric juice is in response to either the mere contact of food with the gastric mucosa, or to distention of the stomach wall. Ivy and Farrell have shown that application of a distending force with the vagi

nerves cut stimulated gastric secretion, hence the secretion cannot be considered "psychic" in origin. The nature of this response is not definitely known. According to Ivy, mechanical distention may stimulate: (*a*) by causing vasodilation and increased motility directly or through intrinsic nerves; (*b*) by stimulating intrinsic secretory nerves which release acetyl choline or histamine or excite the cells by the action potential; (*c*) by direct mechanical stimulation; (*d*) by producing a specific hormone, gastrin; or (*e*) by releasing histamine as the result of mechanical stimulation.

2. Chemical Stimuli.—The second stimulating factor is a chemical or a secretagogue found either in the food or arises from the digestion of food. Experimental work has demonstrated that the stimulus is chemical in nature and is dependent upon substances contained within the food. Meat broths and juices and the products of protein digestion, proteoses and peptones, elicit copious secretion.

Third, Intestinal Phase: It has been shown by a number of investigators that products of gastric digestion upon entering the duodenum act as chemical excitants to gastric secretion. According to Ivy, secretagogues are the only type of stimuli which activate gastric secretion on application to the intestine. Substances which when placed in the intestine stimulate gastric secretion are: meat extracts, water, peptones, soaps, fatty acids (natural fat inhibits), magnesium sulphate, etc. Gastric secretion is inhibited by the presence in the intestine of oil, hydrochloric acid, natural fat, gastric juice, concentrated solutions of sodium chloride, strong solutions of sodium bicarbonate and sugars.

Agents Which Reduce Gastric Secretion.—1. Fats and oils are powerful inhibitors of gastric secretion.

2. Alkalies exert a depressing effect on gastric secretion. This is probably due to a neutralizing action on the acidity rather than to any inhibitory effect.

3. Acid inhibits gastric secretion. When a 1 per cent solution of hydrochloric acid is introduced into the stomach, gastric secretion is completely inhibited. According to Ivy, "acid inhibition" is not observed under all conditions. Acid (0.36 per cent) in the stomach inhibits the action of secretagogues in the stomach (intragastric chemical phase), but not that of secretagogues in the intestine. However, acid in the intestine inhibits the action of secretagogues in both the stomach and intestine. When gastric secretion is stimulated by histamine, acid in the intestine does not inhibit.

4. In man atropine abolishes the continuous secretion of a normal individual, but in the presence of ulcers its action is varied. It does not inhibit the secretagogue action of alcohol and only partially affects the secretory activity of histamine.

5. Section of vagus nerves reduces gastric secretion by eliminating the cephalic phase.

6. Morphine has a mild and brief inhibitory effect.

Agents Which Increase Gastric Secretion.—1. Histamine is one of the most powerful stimulants of gastric secretion. The response to histamine is largely restricted to parietal cells. Because of its selectivity, histamine is used to differentiate between true and false anacidity or achlorhydria. The term achlorhydria is applied to the absence of free HCl acid. This condition may be the result of excessive neutralization, and not of the suppression of acid secretion. Hence, if there is no response after the ordinary test meal, but the glands respond after histamine stimulation the anacidity is characterized as "false." "True" anacidity is a condition in which there is no gastric secretion of free hydrochloric acid after histamine

stimulation. "Achylia gastrica" is a phrase applied when there is complete absence of both acid and pepsin from the gastric contents.

2. Alcohol is another potent stimulant of gastric secretion. It produces a secretion high in acid and mucin. Because of its effect, alcohol was used as a test meal. Various bitters, which are advertised as stomachics, do not have any appreciable effect unless they contain alcohol.

3. Condiments stimulate gastric secretion by stimulating the cephalic phase.

4. Foods. The following foods provoke considerable gastric secretion: juices of vegetables, beef products, chicken, fish, especially salted and fish soups, and legumes containing high protein values.

5. Liver extract used in the treatment of pernicious anemia is also a strong stimulant.

6. Drugs. Insulin, acetyl-choline, pilocarpine, and nicotine are all stimulants of gastric secretion. Insulin administered intravenously produces a hypoglycemic state which stimulates the vagus nerves and induces a secretory response. When the vagus nerves are sectioned the acid response to insulin induced hypoglycemia is abolished. The purpose of this test is to determine if the vagus nerves have been completely sectioned during a vagotomy.

CONSIDERATION OF PEPTIC ULCERS

Etiology.—For want of a better name, gastric and duodenal ulcers have been designated as peptic ulcers, although the term "peptic ulcers" implies that the cause of the ulcers is due to the digestive action of pepsin and hydrochloric acid of the stomach. Dragstedt isolated a Pavlov pouch of the greater curvature and sutured it to a loop of ileum, and observed that gastric juice undiluted by food, saliva, or duodenal contents produced ulcers beyond the anastomosis in all cases. He believes that the major factor in the genesis of ulcer is the acid, hence he concludes that the term "peptic ulcer" should be changed to "acid ulcer."

To date, the actual cause of ulcer is not known, although various theories have been advocated. Almost a century ago Cruveilhier advanced the theory that ulcers were caused by inflammation. He was the first to describe the lesions, and advocated a method of treatment which does not materially differ from the present medical method. Others maintain that gastritis is the etiological factor in the development of peptic ulcers. On the other hand, Walters of the Mayo Clinic contends from his experience that gastritis is a phenomenon of pyloric or duodenal obstruction which is absent when pyloric obstruction is not present.

Because of the nature of its function the stomach is subject to traumatic injury. This is especially true of the pylorus which has been aptly called by the Germans the "magenstrasse." Like all main arteries of communication, this magenstrasse is subject to traffic congestion, and to wear and tear. Deaver called attention to the fact that the acid juices of the stomach are ejected into the duodenum, and impinge against the mucous membrane at a point corresponding to the location of 85 per cent of ulcers. The typical duodenal ulcer is located about 1 inch from the pyloric sphincter, on its anterior or posterior wall. Gastric ulcers generally involve the pyloric part of the stomach, usually on the lesser curvature near the incisura angularis. They are seldom found on the fundus and on the greater curvature, where the acid secreting cells are most numerous. Nor are ulcers found below the ampula of Vater, where the acid chyme is neutralized. Thus it seems justifiable to conclude

that the vast majority of ulcers occur on mucous membrane which secretes an alkaline or neutral fluid under the influence of a hyperacid chyme. Clinical experience has also shown that therapeutic measures, medical or surgical, which aim to overcome hyperacidity and hypermotility bring about the greatest percentage of cures.

Thus, it would appear that acid is an important factor in the genesis of peptic ulcers. However, it must be emphasized that ulcers also occur when the acid content of the stomach is below normal, and even in the presence of anacidity. Apparently a variety of factors are responsible, and the specific cause of the disease has not been determined beyond doubt.

Gastric function is undoubtedly affected by psychic stimulation, although the extent to which it contributes to the formation of an ulcer has not been established. Since 1932, when Cushing first called attention to the incidence of peptic ulcers after intracranial operations, an increasing emphasis has been placed on the role of the psychic phase in the production of peptic ulcers. In 1930 Ivy stressed the importance of pylorospasm due to anxiety, worry, etc., in the genesis of the peptic ulcers. He held the view that pylorospasm produces hemorrhage in the mucosa, which in the presence of digestive juices is converted into erosions or superficial ulcers of variable size and depth depending on the extent of the hemorrhage. The acute lesion does not heal readily because the pylorospasm causes retention. Dragstedt in 1946 editorially emphasizes the role of continuous mental strain, worry and anxiety, and emotions in the pathogenesis of ulcers. These disturbances cause ulcers by producing a hypertonus in the secretory and motor fibers in the vagus nerves. He believes that excessive continuous secretion of pure gastric juice in the fasting stomach is neurogenic in origin and that the physiological basis for the treatment is to eliminate this phase by section of the vagi nerves.

Diseases of adjacent organs, such as the gall bladder and the appendix, are factors favoring the development of ulcers. For instance, peritoneal irritation from any cause will produce pylorospasm accompanied by gastric hypersecretion, thus accounting for the fact that diseases of the appendix or gallbladder are not uncommonly associated with peptic ulcers. Other etiological factors to consider are: vascular insufficiency, specific bacterial infection, toxic causes, etc.

Indications for Surgical Treatment of Peptic Ulcers.—Considering the fact that the physiologist is still seeking for the etiological factor or factors in the genesis of ulcers, it is inevitable that a wide difference of opinion should exist as to the treatment. Until such time when the causative agent has been found, ulcers must be treated empirically and unbiased by personal prejudices or vagaries.

Whether to treat peptic ulcers medically or surgically had been for years the bone of contention between the internist and the surgeon. We have had the biased clinician who held fast to the tenet that the ulcer problem was a medical one; while on the other hand, we have had the equally self-opinionated surgeon who enthusiastically maintained the view that surgery offered the solution of the problem. Incidentally, both have made extravagant and unwarranted claims as to the results that were obtained. However, it is worthy of note that within the past two decades this attitude has been changing to the extent that at the present time a spirit of coöperation is the rule rather than the exception.

The decision to operate should be based upon a careful consideration of the history, and a careful analysis of the physical and laboratory findings of each individual case, for each case presents a different and distinct problem. Further-

more, a thorough knowledge of the various phases of gastroduodenal ulcers is essential before assuming the responsibility of deciding whether surgery is indicated. This includes an understanding of the prevalent theories of the etiology, a knowledge of the pathology, pathogenesis, symptomology, and known results of both medical and surgical treatment of peptic ulcers.

There is universal agreement about the advisability of thorough medical treatment before subjecting a patient with peptic ulcer to surgery. The basic principles in ulcer therapy, whether medical or surgical, consist of overcoming pylorospasm, reducing hyperacidity, diminishing gastric hypersecretion, allowing the stomach to empty in a normal or quicker than normal time, and placing the stomach at physiological rest. Medical treatment includes the use of anti-spasmodics to overcome pylorospasm; control of hyperacidity with antacids; control of hypersecretion with selective foods and proper hygiene; and giving rest to the stomach with small frequent feedings. When these measures are intelligently adhered to, ulcers will heal, or at least a recession of the symptoms will occur in the great majority of uncomplicated peptic ulcers.

Surgery is indicated when complications of peptic ulcers develop which are not amenable to adequate medical therapy, or the complication, *per se*, may jeopardize the patient's life. These complications may be classified as follows:

1. Obstructive lesions producing motor dysfunction of the pylorus:
   a. Those due to scar tissue.
   b. Those due to edema and pylorospasm.
2. Perforation:
   a. Acute perforation.
   b. Sub-acute.
   c. Chronic perforation.
3. Hemorrhage
4. Failure of repeated medical treatment to relieve the symptoms.
5. Gastric ulcer associated with carcinoma.

One of the most frequent complications of peptic ulcer is pyloric obstruction which may occur as a result of cicatricial stenosis, inflammatory edema, or spasm. Patients with cicatricial obstruction will give a history of having had recurrent attacks covering a period of years. These attacks are due to recurrent ulcerations which produce various degrees of motor-dysfunction as a result of multiple cicatrix. If the scars are located close to the pyloric sphincter, the patient will complain of symptoms of pyloric obstruction, the severity of which will depend upon the degree of cicatrization. Because of the nature of this complication, medical therapy is of no avail. We are dealing here with a motor mechanical defect, and a short-circuiting operation, such as a gastrojejunostomy will produce immediate relief.

Obstruction due to edema and spasm is generally of sudden onset, accompanied by vomiting and by marked aggravation of pain. The treatment, which is primarily medical, consists of keeping the stomach empty by continuous suction with a duodenal tube, and measures to overcome pylorospasm. Supportive treatment with intravenous infusions of amino acids, glucose and saline is also indicated. This treatment generally relieves the pyloric obstruction; however, if the symptoms persist, or if they recur, surgical interference may be necessary.

In the acute perforating ulcer, surgery is a lifesaving measure. We are dealing here with an acute surgical abdomen when time is an essential element, because the

chances for the patient's recovery decreases with the delay in the operation. The patient's chances are extremely good if he is operated upon within the first six hours; whereas, after the first twelve hours the mortality rate is about 10 per cent and higher after the first twenty-four hours. The primary consideration in the operative treatment is the closure of the perforation. Additional procedures such as gastrojejunostomy, pyloroplasty, or gastric resection at the time of the original operation is not justified and only adds to operative risk. Simple closure with suture and omental graft reinforcement gives good results. If necessary a secondary operation, if indicated, may be done at some future date. In cases where adequate closure cannot be obtained without producing pyloric occlusion, a posterior gastrojejunostomy or any of the pyloroplasties is indicated.

Surgery is also indicated in an "impending perforating ulcer." The diagnostic feature is persistency of pain even though the patient is under strict medical treatment. This is due to the fact that the ulcer has eroded through all the layers of the gastric wall with the exception of the serosa. Medical treatment will not prevent perforation; hence surgery is done to forestall an acute rupture of the ulcer. The operative procedure consists of excision of the ulcer by a V-shape section or by partial gastrectomy. The latter procedure is preferred. This type of ulcer should be classified as a subacute perforation.

Singer and Vaughan, of Chicago, described another type of perforation which they called "Formes frustes." This type of perforation, although ushered in by symptoms of a perforate peritonitis, fails to develop evidences of diffusely progressive peritonitis as in the acute perforations, but, instead, produces rather mild symptoms which abate quickly. The spontaneous recovery occurs from sealing or plugging of the perforation, and as a consequence there is only a limited escape of gastric juices or duodenal content. The peritoneum is readily able to cope with the relatively sterile and small amount of foreign liquid, therefore only a slight peritoneal disturbance ensues. "Formes frustes" perforations are not uncommon. They are frequently overlooked as a severe gastric episode by the patient or physician; or they are diagnosed as acute perforations, acute appendicitis, etc. If the condition is recognized within the first twenty-four hours, the author advises a surgical operation regardless of the severity or mildness of the symptoms because one is not able to determine the true condition from the symptoms and physical findings alone. After the first twenty-four hours if the signs and symptoms point indubitably to a spontaneous closure, surgical treatment is not instituted. However, if there are signs of peritoneal irritation indicating an open perforation there should be no hesitation to operate.

A frequent complication of duodenal ulcer is one which has eroded into the pancreas. This is an example of a chronic perforated ulcer. The patient usually gives an atypical history of duodenal ulcer which may date back over a period of years. The relief by food or alkalies is not constant. Because of the pancreatic involvement, pain in the back and loss of weight may be so extreme as to obscure the diagnosis. When the perforation into the pancreas actually occurs pain may be severe enough to resemble an acute inflammatory process; however symptoms of peritoneal irritation are absent, and the patient will give a previous history of peptic ulcers. A chronic perforating duodenal ulcer is extremely intractable to medical management. It constitutes one of the most important indications for surgical treatment. The type of procedure will depend on the condition of the ulcer at the operation. If the defect is small and has not penetrated the pancreas, the

ulcer slit may be excised according to the method of Balfour. For a large ulcer or one which has penetrated the pancreas, a partial gastrectomy is the ideal procedure. To avoid acute necrosis of the pancreas the ulcer base may be left on the pancreas by carefully excising the margins of the ulcer; or, better, the resection may be done above the ulcer. In the latter instance the ulcer heals promptly because the acid gastric contents are excluded from the duodenum.

Hemorrhage from a peptic ulcer is a serious complication which not infrequently has a fatal termination. In deciding the question of medical or surgical treatment, there are many factors to be considered. Hemorrhage occurring in a young patient should be treated expectantly, because such hemorrhage is rarely fatal. We believe that the patient should be operated upon when he has recovered from the effects of the hemorrhage. There are surgeons who prefer to operate after a second hemorrhage. This is dangerous, since experience teaches that recurrent hemorrhages are more severe and more likely to terminate fatally. Immediate operation for hemorrhagic ulcers should be considered in patients past the age of forty especially those with arteriosclerotic vessels. If an immediate operation is undertaken it should be done early, within the first twenty-four hours, or a fatality will result even though blood transfusions have been given. The author has found that when a patient requires more than one transfusion, he is a candidate for surgery. The type of operation will depend upon the site of the ulcer, condition of the patient and whether it is an emergency measure. In the latter instance, the simplest procedure that will stop the hemorrhage should be done. This may be accomplished either by ligating the vessels outside of the stomach wall, or by suturing the bleeding ulcer through a gastrotomy.

Another surgical indication is an ulcer of the stomach having potential malignant possibilities. However this is not true of duodenal ulcers as they are rarely if ever malignant. It has been definitely established that there are gastric ulcers which have all the characteristics of benign lesions, yet prove to be malignant. Whether an ulcer is a malignancy in its inception, or a benign ulcer which develops carcinomatous changes, is a question which cannot be answered at the present time. In view of the fact that about 20 per cent of all carcinomas occur in the stomach, it is entirely feasible that benign ulcer and carcinoma of the stomach may be found concommitantly. Regardless of the etiological relationship, it is known that from 10 to 20 per cent of gastric ulcers eventually prove to be malignant lesions and that the symptoms, physical findings, *x*-ray examination, etc., are inadequate in differentiating the two conditions. However, there are certain features of the malignant ulcer which assist in the differentiation. The following criteria are of value in [illegible]

[illegible] until the test of time proves it to be benign. It is important to bear in mind that a patient with a malignant ulcer may respond symptomatically under medical treatment; even the *x*-ray niche may show a reduction in size. For this reason, these individuals should be under constant observation for long periods of time, and whenever the ulcer recurs, there should be no further procrastination in deciding to operate.

2. Ulcers located within 1 inch of the pylorus and those on the greater curvature should be considered malignant and should be treated surgically regardless of all other factors.

3. Radiological evidence of an ulcer defect over 1 inch in diameter is most

likely to be a malignant ulcer. This is especially true if the size of the niche fails to reduce after three weeks of medical treatment.

4. Ulcers which fail to respond to medical therapy as evidenced by persistence in the symptoms and increase in size of the niche should be considered as malignant ulcers and early surgery advised.

5. The strong possibility of malignancy exists when the gastric analysis shows achlorhydria or achylia. However, it is important to realize that some cases of carcinoma have acidity values approximating the normal, and some cases of benign gastric ulcer have anacidity. Just because a gastric analysis shows high values for acid does not necessarily exclude the probability of carcinoma of the stomach. In such cases careful observation under medical treatment with frequent *x*-ray examinations will disclose the true nature of the lesion. Achlorhydria is undoubtedly the best evidence of malignancy and if other diagnostic methods and physical findings corroborate the diagnosis, malignancy is practically certain.

Physiological Basis for Surgical Treatment of Peptic Ulcers.—The basic principles upon which the philosophy of nearly all operative procedures in the treatment of peptic ulcers are: (1) to save life; (2) to relieve the patient from disabling complications; (3) to remove a potential malignant ulcer; and (4) to alter the maladjustment in the gastric physiology so that the procedure is attended with a minimum of chance for a postoperative ulceration.

Of the many methods advocated there are four cardinal procedures, each having certain advantages and disadvantages. These are: (1) Short-circuiting operations such as gastrojejunostomy; (2) methods which aim to remove the ulcer followed with pyloroplasties; (3) vagotomy; and (4) partial gastrectomy.

It may seem trite to emphasize that no one method will satisfy the requirements of every condition, and that the procedure of choice should be based on the selective needs of each individual case wherein its application will carry out the objectives of the four cardinal basic principles in the surgical treatment of peptic ulcers. The surgeon should bear in mind that unless the exigencies of the case demands it, he should not superimpose on the patient the added risk of an unnecessary reconstructive procedure. For instance, it is not conducive to good surgery to suture a perforated peptic ulcer and then proceed to perform a gastrojejunostomy upon a patient who has had as much as he can stand. Simple closure is the primary requirement in this case in order to save life; any reconstructive operation should be done only if this additional procedure is essential to recovery. The surgeon should be careful not to neglect the physical status of the patient. Many young adults are physiologically older than their actual age, therefore greater operative risks. A young adult with a cardio-renal disease may not withstand anything but the simplest palliative procedure. A patient in this category may have a resectable gastric lesion, but his physical condition would not permit a gastric resection. The surgeon must be satisfied to do a lesser procedure in this case. It is wise to carefully evaluate the constitutional fitness of each patient and judiciously select the procedure which will accomplish its objective without undue risk to the patient.

1. **Short Circuiting Procedures.**—In former years a gastrojejunostomy was largely favored among surgeons in the treatment of peptic ulcers mainly because of the low mortality rate which attended this operation. However, it was soon learned that a more formidable procedure, such as gastric resection, does not carry any higher risk. Furthermore, the surgeon should bear in mind that he is not operating for a low mortality rate, but because the patient presents a problem which must

be dealt with according to the findings at operation. A gastrojejunostomy is primarily a procedure to relieve pyloric obstruction. If pyloric obstruction is the complicating factor in the patient's complaint, then this operation will give excellent results. However, if the pylorus is not obstructed, the results are not satisfactory. It is undoubtedly the procedure of choice in cases of acute pylorospasm which fail to respond to medical therapy; in the old chronic ulcer with cicatricial contraction of the pyloric sphincter; and in all cases which are accompanied by mechanical motor-dysfunction of the stomach. It is generally agreed that gastrojejunostomy is not the operation of choice in cases where persistent high acid content does not respond to medical treatment.

A gastrojejunostomy, if performed when adequate indications exist, brings about relief of symptoms and healing of the ulcer because of one or more of the following factors:

a. It decreases the emptying time of the stomach. Pyloric stenosis prolongs the gastric phase of secretion, thus a gastrojejunostomy removes this factor by decreasing the emptying time.

b. It reduces the gastric hyperacidity by mixing the alkaline duodenal contents with the gastric juices through the new opening and by decreasing the emptying time.

c. It provides rest to the stomach

d. It by-passes the acid juice and chyme from a duodenal ulcer.

2. **Pyloroplasty.**—Numerous plastic procedures have been devised for the surgical treatment of bleeding and penetrating type of duodenal ulcers. It is generally agreed that the bleeding type of ulcer should be excised if permanent control of bleeding is desired. This may be accomplished by any one of the following methods: (1) excision of the ulcer by cautery combined with gastrojejunostomy; (2) excision by cautery combined with pyloroplasty; (3) excision by knife combined with pyloroplasty according to the method of Finney, Balfour, Horsley, or Heineke-Mikulicz; (4) pylorectomy, such as Bilroth 1 method; and (5) partial gastrectomy.

Pyloroplastic methods combined with local excision of the ulcer is seldom done at the present time, because experience has shown that these methods do not alter the disturbance in gastric secretion and that recurrent ulcers are frequent. The procedure of choice is partial gastrectomy. When the condition of the patient does not permit a gastric resection, local excision of the ulcer combined with either Finney's pyloroplasty or posterior gastrojejunostomy is done.

3. **Vagotomy.**—Recent years has seen increasing emphasis on the role of the cephalic phase of gastric secretion as a factor in peptic ulcers. Since this phase of gastric secretion is under the influence of the vagus nerves it is logical that methods should be devised to interrupt the vagal influence on the stomach. Experimental and clinical observations have demonstrated that by sectioning the vagi the cephalic phase is annuled thereby reducing the total secretion of gastric juice induced by a meal. Dragstedt and others are utilizing this principle in the treatment of peptic ulcers, and their reports indicate that psychomatic factors play a dominant role in the pathogenesis of ulcers. According to Dragstedt both the hypermotility and hypersecretion, which occur in the empty stomach in the absence of the usual stimulus of food, are abolished by complete section of the vagus nerves. He found that patients are relieved of the usual gastric distress and that this relief is not due to interruption of sensory fibers is indicated by the fact that the symptoms can be reproduced again by the installation of a solution of hydrochloric acid into

37. What is the effect of the following on gastric secretion: alcohol; vegetable juices; beef products; liver extract; insulin; nicotine; and pilocarpine?
38. What is the purpose of the insulin test?
39. Discuss the etiology of peptic ulcers.
40. Which part of the stomach is usually affected with ulcers?
41. Discuss the role of acid in the pathogenesis of ulcers.
42. Discuss the role of neurogenic factor in peptic ulcers.
43. Discuss the surgical indications for peptic ulcers.
44. How would you manage pyloric obstruction due to scars and that due to edema and spasm?
45. Would you perform a gastrojejunostomy after closing an acute perforated peptic ulcer? Explain.
46. What is an impending perforated ulcer?
47. Define: "Formes frustes" perforation.
48. How would you treat this type of ulcer if diagnosed in twenty-four hours? Forty-eight hours?
49. Discuss the symptoms and differential diagnosis of an ulcer which has penetrated into the pancreas.
50. How would you treat this type of ulcer?
51. Discuss the management of bleeding ulcers.
52. Discuss the relationship of gastric ulcer and carcinoma.
53. Give the criteria in determining the probable malignancy of an ulcer.
54. What are the basic principles of operative procedures in the treatment of peptic ulcers.
55. Discuss the physiological basis of gastrojejunostomy in the treatment of peptic ulcers.
57. Discuss the role of vagotomy in the treatment of peptic ulcers.
58. Upon what three factors is the efficacy of vagotomy dependent upon?
59. Discuss the physiological basis of partial gastrectomy in the treatment of peptic ulcers.

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CHAPTER 50

ANATOMY OF THE STOMACH

By A. V. Partipilo

Anatomical Relationship.—The greater part of the stomach lies within the left half of the epigastric and in the left hypochondriac zones. The main portion of the stomach lies on the left side of the body. Its shape and position varies considerably and depends on the volume of the contents of the stomach. The normal stomach also varies according to individual body habitus and its form and position may be of three types: (1) The high transverse type is seen in the hypersthenic bodily habitus. The transverse position of the stomach is due to the large amount of fat in the abdomen; the stomach is pushed under the diaphragm and the pylorus often lies to the right of the body of the spine. (2) The cow-horn type of stomach is seen in the average type of individual with asthenic bodily habitus. The stomach is crescent shape, the fundus reaches the level of the umbilicus and the pylorus is usually found at the midline. (3) The third type is the fish-hook stomach which is seen in the thin asthenic type of individuals. The fundus is usually found in the pelvis and the pylorus is to the left of the spine. The emptying time of this type of stomach is markedly delayed. When fully distended it comes in contact with the inferior border of the left lobe of the liver medially, and laterally with the diaphragm. Below, its wider anterior surface comes in contact with the anterior abdominal wall. The posterior surface rests on the spleen, left kidney, transverse colon and the pancreas (Fig. 326).

The stomach is the connecting organ between the esophagus above and the duodenum below. Hence, its most fixed portion is at the cardia where it becomes continuous with the esophagus, and at its terminal portion where it ends in the duodenum.

Attachments of the Stomach.—The stomach is wholly within a peritoneal covering. On its lesser curvature is attached the lesser omentum, called the gastro-hepatic ligament, which connects the lesser curvature of the stomach to the inferior surface of the liver. That portion of the lesser omentum passing from the porta hepatis of the liver to the superior part of the duodenum is called the hepato-duodenal ligament. The lesser omentum contains the right and left gastric arteries, lymph glands and branches of the vagus nerve. The hepato-duodenal ligament forms the anterior layer of the foramen of Winslow within which are located the common duct, hepatic artery, portal vein, and lymphatic vessels (Fig. 327). The gastrocolic omentum connects the stomach with the transverse colon, and is prolonged as the great omentum. The gastrocolic omentum contains the right and left gastric epiploic vessels. The fundus and part of the body of the stomach is attached to the hilum of the spleen by a double layer of peritoneum called the gastro-splenic ligament. This ligament is continuous with the gastro-colic ligament. It contains the short gastric vessels, vasa brevæ, derived from the splenic artery. Finally, a thin fold of peritoneum passes from the stomach along the left side of the esophagus

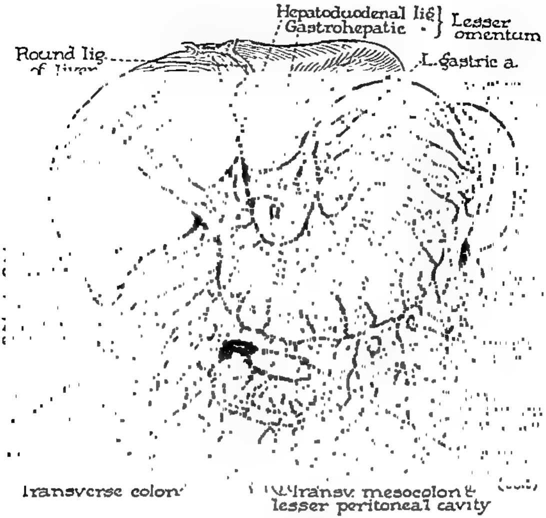

Fig. 326.—Anatomical relationship of the stomach.

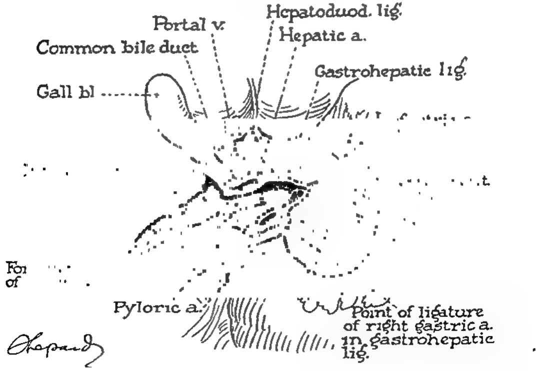

Fig. 327.—Structures anterior to the foramen of Winslow.

to the diaphragm. This is called the gastro-phrenic ligament which aids in the mobilization of the upper end of the stomach and esophagus.

Divisions of the Stomach.—The esophagus, after passing through the esophageal hiatus of the diaphragm, opens into the cardiac portion of the stomach at the level of the tenth or twelfth thoracic vertebra. The cardia is deeply situated at the superior end of the lesser curvature, and to its left is a sac-like portion which projects higher up. This is the rounded, dome-shaped fundus. The fundus may be said to consist of that portion of the stomach lying above a horizontal plane drawn from the esophageal opening (Fig. 328). The middle and largest part of the stomach is the body or corpus. It extends from the fundus to the incisura angularis on the lesser curvature, and to a notch on the greater curvature which marks the beginning of the pyloric antrum. The pylorus begins at the incisura angularis and is continued into the duodenum. The pylorus is usually cylindrical in shape and is divided into

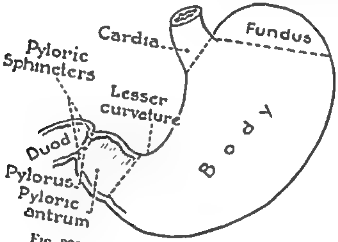

Fig. 328.—Divisions of the stomach.

the pyloric canal, and pyloric antrum. It is separated from the duodenum by a ring-like constriction, the pyloric sphincter. This ring-like projection serves as a guide in distinguishing the pylorus from the duodenum. Another guide is the pyloric vein of Mayo which is seen crossing the bowel at the junction of the duodenum and the pylorus.

Blood Supply of the Stomach.—The stomach receives its blood supply from the coeliac axis, a short but wide vessel arising from the abdominal aorta about 1 centimeter below the aortic orifice of the diaphragm (Fig 329). It lies between the two crura of the diaphragm in the region of the first lumbar vertebra, behind the omental bursa where it runs forward for 12 millimeters between the caudate lobe of the liver above and the upper border of the pancreas and the splenic vein below. Uncontrollable bleeding from the stomach can be stopped by making pressure on the coeliac axis. The latter can be found by passing the hand over the caudate lobe of the liver to its junction with the body of the vertebra. The coeliac axis will be found just below this junction. By alternate pressing and releasing of the artery, the bleeding point will be located, grasped in a forceps, and ligated. The coeliac axis terminates by dividing into three branches: (1) hepatic artery; (2) left gastric artery; and (3) splenic artery.

1. The hepatic artery runs along the upper border of the pancreas between the layers of the gast-o-pancreatic fold to reach the first part of the duodenum. From

there, it passes upward between the hepatico-duodenal ligament anterior to the portal vein, and to the left of the common bile duct to reach the liver. From the hepatic arise the right gastric and the gastro-duodenal arteries. The right gastric artery springs from the hepatic within the gastro-hepatic ligament just before the hepatic begins to turn upward. It then descends between the two layers of the duodeno-hepatic ligament, and when it reaches the duodenum, it turns to the left to supply the pyloric and lesser curvature side of the stomach. It ends by anastomosing with branches of the left gastric artery in the region of the incisura angularis.

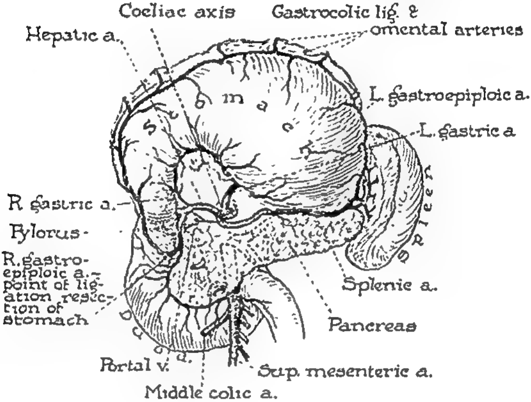

FIG. 329.—Blood supply to the stomach.

When performing a partial gastrectomy, the right gastric artery is ligated within the duodenal hepatic ligament. Because of the soft character of the tissues within which the artery is imbedded, it will instantly retract when severed. For this reason, when ligating, make certain that the ligature is tied securely. Should the ligature slip during the operation, the method of locating the bleeding point is to insert the index finger into the foramen of Winslow and the thumb anteriorly to the duodenal hepatic ligament; then by alternately pressing and releasing the hepatic artery, the bleeding point is determined, and the divided vessel is found and religated.

The gastroduodenal artery arises from the hepatic at a point where the duodenum descends. From this point, its course is downward behind the duodenum, where it divides into the right gastro-epiploic and the duodeno-pancreatic arteries. The right gastro-epiploic artery passes to the left between the gastro-colic omentum along the greater curvature of the stomach, to supply the pylorus. It anastomosis with the left gastro-epiploic artery at the junction of the body and the pyloric antrum. The pancreatico-duodenal artery runs for a short distance between the head of the pancreas and the first part of the duodenum, finally dividing into the superior

duodenal and superior pancreatic arteries. These eventually anastomose with similar branches of the inferior pancreatico-duodenal vessels arising from the superior mesenteric artery.

2. The left gastric artery is the smallest branch of the coeliac group of vessels; however, it is the largest of the arteries supplying the stomach. From its point of origin, the artery runs upward and to the left behind the omental bursa, reaching the lesser curvature close to the cardia. After giving off esophageal branches, it divides into two branches which run along on either side of the lesser curvature and anastomose with branches of the right gastric artery. The best approach to the vessels is through the gastro-colic ligament.

3. The splenic artery is the largest of the coeliac branches. It has a tortuous course, traveling behind the stomach and the omental bursa, along the upper border of the pancreas, then passing forward between the two layers of the lieno-renal ligament when the branches to the stomach pass onward between the gastro-splenic ligament. It gives off four or five short branches (vasa brevia) to the fundus and the left gastro-epiploic which gives off branches to the body of the stomach. The latter vessel passes along the greater curvature between the layers of the gastro-colic omentum, and terminates by anastomosing with the right gastro-epiploic artery.

Lymphatic Glands of the Stomach.—Accurate knowledge of the lymphatic system of the abdomen is essential if the operative treatment of gastric malignant disease is to be successful. The lymph glands of the abdomen are divided into visceral and parietal groups. The visceral groups are those associated with the lymph vessels, which in general follow the principal visceral branches of the aorta. They lie between the layers of the various omenta, ligaments, and mesenteries. The visceral lymph nodes may be considered as being primary, since they receive lymph vessels from the viscera. The parietal glands are found closely associated with the abdominal part of the aorta, the inferior vena cava, and the adjacent parts of the posterior abdominal wall. The parietal nodes may be classified into a coeliac group found in the vicinity of the coeliac axis and the superior mesenteric artery; and a lumbar group consisting of three or more rows of glands along the course of the abdominal aorta from the second lumbar vertebra to the aortic bifurcation. Lymph glands along the splenic, hepatic, and superior mesenteric artery, communicate by their efferents mainly with the coeliac nodes; while those along the inferior mesenteric artery communicate with the lumbar group of nodes. The coeliac nodes, 15 to 20 in number, receive lymphatic drainage from the liver, spleen, pancreas and stomach. They drain into the cisterna chyli by way of the main intestinal lymph trunk.

Anatomical textbooks do not agree in the terminology nor in the classification of the lymph glands of the stomach. From a surgical point of view they may be classified into six groups as follows:

1. *Cardiac Group.*—In this group are found the right, left, and the posterior paracardial lymph nodes. The right paracardial nodes are situated to the right of the cardiac orifice, from which they receive afferent branches. They send efferents to the posterior left gastric nodes. The left paracardial nodes lie to the left of the esophageal orifice, and receive afferents from the adjacent part of the stomach. They send efferents to the posterior left gastric nodes (Fig. 330).

2. *Left Gastric Nodes.*—These follow the course of the left gastric artery, and are situated between the two layers of the lesser omentum. They receive afferents

from the anterior and posterior surfaces of the lesser curvature. They send efferents to the nodes along the hepatic artery and to the middle supra-pancreatic lymph glands which lie around the coeliac axis. Those glands along the main stem of the left gastric artery are known as the posterior left gastric lymph nodes, whereas those along the branches of the artery in close proximity with the stomach are known as the anterior left gastric nodes.

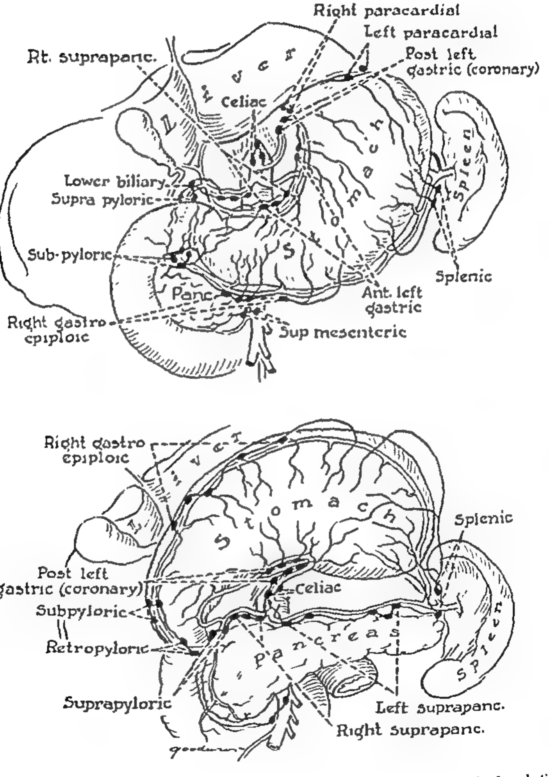

FIG. 330 —Lymph glands of the stomach. (*a*) anterior view; (*b*) posterior view showing lymphatic groups along the hepatic and splenic arteries.

CHAPTER 51

GASTRIC SURGERY—GASTROJEJUNOSTOMY

By A. V. Partipilo

TECHNIQUE

Through a paramedian or Sloan's incision, the abdomen is opened, and the stomach, including other abdominal organs, is examined. The transverse colon is found, and pulled out of the abdominal incision. Note the position of the mid-colic artery, and the length of the mesocolon. A posterior gastrojejunostomy cannot be performed if the mesocolon is too short, or if the mid-colic artery forms a short arch at the base of the mesocolon.

First Loop of Jejunum.—When the terminal duodenum reaches a point opposite the left side of the first or second lumbar verterbra, it turns abruptly forwards, downwards, and to the left, and passes into the jejunum (Fig. 332). This bend, known as the duodenal-jejunal flexure, is fixed by a thin band of unstriped muscle, which is attached to the left crus of the diaphragm. This band is known as the ligament of Treitz. Its relative length determines the position and length of the flexure. If the ligament is short (Fig. 332), the flexure is placed rather high, thus predisposing to acute angulation and kinking. The condition may be accentuated after a gastrojejunostomy, especially when the patient is in the upright position. For this reason the surgeon should examine the duodenal-jejunal flexure, and if found acutely angulated, the ligament of Treitz should be divided and its ends ligated.

To locate the first loop of jejunum, the transverse colon is grasped with the left hand and thrown upwards, as illustrated in Figure 333. Now pass the right hand along the taut mesocolon until the spine is reached. The index finger is flexed, and the middle finger is made to follow the mesocolon to the left of the body of the vertebra in a direction toward the inferior body of the pancreas. The middle finger is then flexed, thus hooking the first loop of jejunum between the index and middle fingers (Fig. 333). The proximal end of the first loop of jejunum is relatively fixed by the ligament of Treitz, and for this reason when traction is applied upon the loop within the index and middle fingers, it will not be possible to draw out any bowel from the cephalad end. On the other hand, if the proximal end of the bowel within the grasp of the fingers can be milked out, the segment is not the first loop of jejunum. Another method of locating the first loop of jejunum is to expose the mesocolon at its junction with the body of the vertebræ. The terminal portion of the duodenum, after ascending to the top of the second lumbar vertebra, ends abruptly and becomes the *jejunum* by emerging from the retroperitoneal space through a reflection of the transverse mesocolon (Fig. 332, *a*). Thus, in order to locate the first loop of jejunum it is necessary to expose the mesocolon at the junction of the vertebra where the mesentery is reflected on the jejunum. This is accomplished by pulling the transverse mesocolon taut until its attachment

The glandular groups which should be removed with a carcinoma of the stomach are:

1. The right paracardial lymph nodes found at the right cardiac orifice of the stomach.

2. Left gastric nodes on the lesser curvature. These glands communicate freely with the above group and if involved the right paracardial glands are very likely involved also.

3. Posterior left gastric glands are found along the left gastric artery. They constitute a most important group of gland and must be removed entirely. For this reason the left gastric artery is ligated and divided close to its origin at the coeliac axis. These nodes, with the artery, are then removed en masse with the left gastric and right paracardial groups of nodes.

4. The right and left gastro-epiploic nodes are removed by dividing the gastrocolic omentum close to the transverse colon.

5. The pyloric groups are also carefully removed and if any large glands are found along the supra-pancreatic group, they are also excised.

6. The splenic group of lymph glands are rarely involved because of the infrequent involvement of the fundus of the stomach with malignancy. However, if this portion of the stomach is primarily affected with carcinoma, then it is necessary to remove the splenic group of glands with the spleen.

QUESTIONNAIRE

1. In what type of individual is the high transverse stomach found?
2. What is meant by: (a) cow-horn stomach, (b) fish-hook stomach?
3. Describe the formation of the lesser and greater omenta.
4. What vessels are found within the lesser omentum?
5. Describe the various omental and ligamentous attachments of the stomach.
6. Give the various anatomical divisions of the stomach.
7. How would you determine the junction of the pylorus and the duodenum?
8. Give the relationships and location of the coeliac axis.
9. Give a practical method of locating it.
10. Give the relation, branches, and distribution of the following: (a) Hepatic artery. (b) Left gastric artery. (c) Splenic artery.
11. What precaution should be taken when ligating the right gastric artery?
12. Draw a diagram showing the blood supply of the stomach.
13. Which of the coeliac vessels is the largest artery?
14. Which gastric artery is the largest?
15. Discuss the lymph drainage of the abdomen.
16. Give the main groups of lymph glands which directly receive lymph drainage from the stomach.
17. What groups of glands are removed with a malignant growth of the stomach?

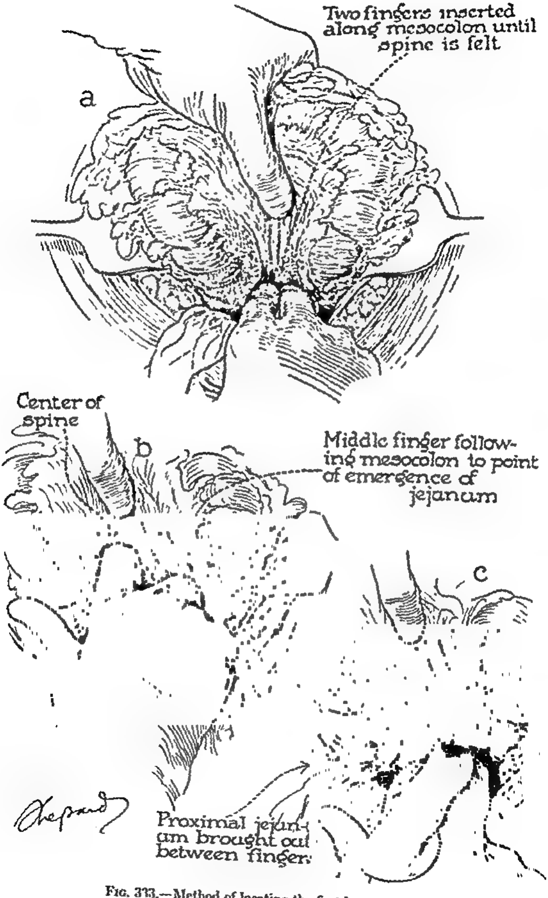

FIG. 333.—Method of locating the first loop of jejunum.

to the spine is seen. The first loop of jejunum will be seen to emerge at the second lumbar verterbra.

After the first loop of jejunum has been found, its positions and direction should be noted. Figure 334 illustrates the various positions of the loop in relation to the spine. In about 85 per cent of cases it is directed to the left and downward; in others, it may be longitudinally downward, and rarely, it may be directed to the right, across the spine.

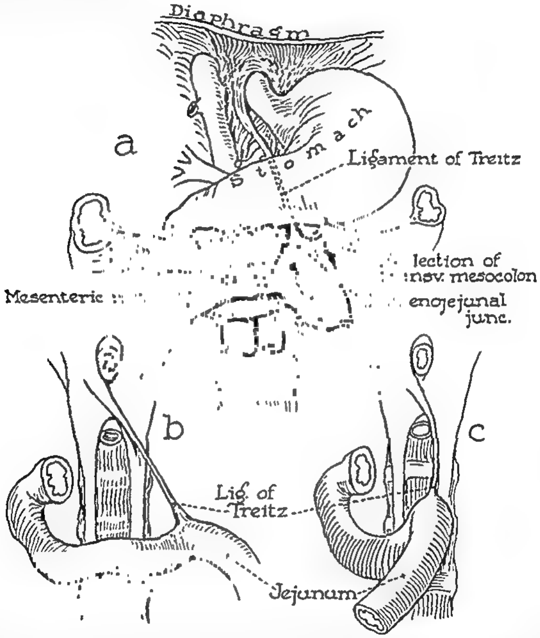

FIG. 332.—(a) Schematic representation of the duodenum and its retroperitoneal relationship; (b) elongated ligament of Treitz allowing the duodenaljejunal flexure to lie below the transverse mesocolon; (c) represents the U-type of duodenum with the ligament of Treitz elevating the flexure above the transverse mesocolon thus producing an acute angulation

Types of Anastomoses.—After having observed the position of the first loop of jejunum, and the relationship of the mesocolon and mid-colic vessels, the next step is to decide where the opening is to be made on the posterior surface of the stomach. The gastro-enterostomy opening must be made in the most dependent portion of the stomach, close to the curvature. This point is opposite the incisura angularis,

and will be crossed by a line dropped from the right side of the esophageal orifice. A line drawn from this point, direct to the right toward the lesser curvature, and parallel to the loop of jejunum, will outline the proposed opening in the stomach. In this manner, after the jejunum is sutured to the stomach, the normal position of the loop is not altered. The jejunum is anastomosed to the stomach so that its proximal end is approximated *close to the lesser curvature*, whereas its distal end is close to the greater curvature. When the position of the jejunum is straight downward, the incision on the posterior surface of the stomach is made from the lesser to the greater curvature parallel to the direction of the loop. When the loop normally

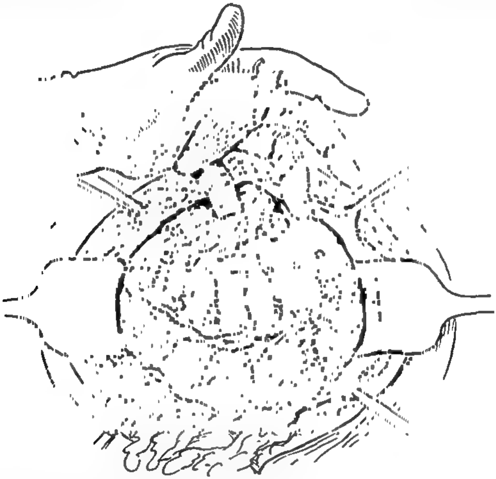

Fig. 336.—Author's method of outlining the gastric incision on the anterior surface of the stomach. The little finger is placed at the acute angle of the stomach.

lies across the spine, the incision is made parallel with the greater curvature. In this instance the proximal end of the jejunum is approximated to the cardiac end, and the distal to the pyloric end of the incision (Fig. 334).

The above methods of gastroenterostomy are anatomical, antiperistaltic anastomoses, except when the opening is made parallel with the greater curvature, the anastomosis is not only anatomical but also physiological. There are surgeons who prefer *the isoperistaltic, physiological anastomosis* instead of the anatomical. This is done by making the incision on *the posterior surface of the stomach parallel* with the greater curvature. The proximal end of the jejunum is approximated to the cardiac end, and its distal end to the pyloric end. In this manner the peristaltic waves of both the stomach and the jejunum will travel in the same direction.

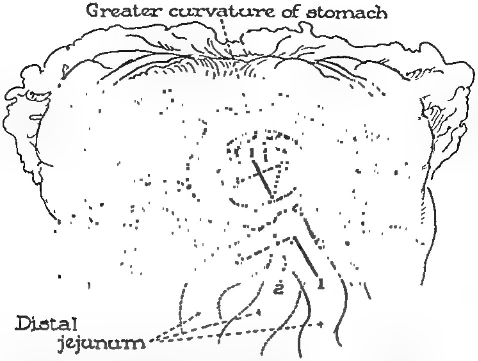

FIG. 334.—Location and direction of incisions in the stomach and jejunum for posterior gastrojejunostomy; (1) from the lesser curvature to the greater curvature according to the most common direction of the first loop of jejunum; (2) from the lesser curvature to the greater curvature when the first loop of jejunum is found vertically downward; (3) isoperistaltic anastomosis.

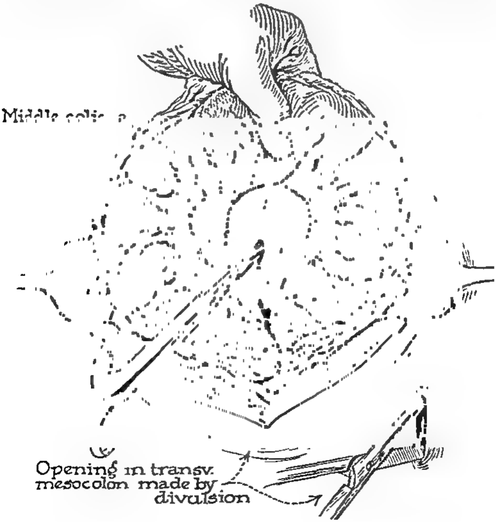

FIG. 335.—With scissors a blunt incision is made in the mesocolon to the left of the mid-colic artery.

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Delivery of the Stomach Through the Mesocolon.—After having decided the type of anastomosis, lift up the transverse colon, and by traction the mesocolon is exposed and made taut. As illustrated in Figure 335 a blunt incision is made in an avascular area of the mesocolon to the left of the mid-colic artery. The surgeon should avoid injuring this vessel.

While the assistant pulls the colon taut, the operator outlines the incision by hooking the incisura angularis with the little finger of the left hand (Fig. 336),

FIG. 337—Author's method of outlining the incision. Note the position of the little finger at the incisura angularis and the index finger at the greater curvature. Allis forceps are passed through the mesocolic rent grasping the posterior surface of the stomach corresponding to the points beneath the little and index fingers.

and the index finger is placed on the low point on the greater curvature. As illustrated in Figure 337, a line between the two fingers outlines the proposed incision on the posterior surface of the stomach. Pass Allis forceps through the slit in the mesocolon, and grasp the posterior wall of the stomach corresponding to the points indicated by the index and little fingers (see inset of Fig. 337). By downward traction the posterior surface of the stomach is delivered through the mesocolic rent. Thus, with this method, only that part of the stomach which is to be used for the anastomosis is presented. The line between the two Allis forceps

corresponds to the line of incision to be made. This method of delivering the stomach eliminates guesswork and avoids unnecessary manipulation.

Two mattress stitches are placed to fasten the mesocolic edge to the lesser curvature of the stomach (Fig. 338). The remaining mesocolic edge is left loose. Next, place about three inches of the stomach in a flexible intestinal clamp. The

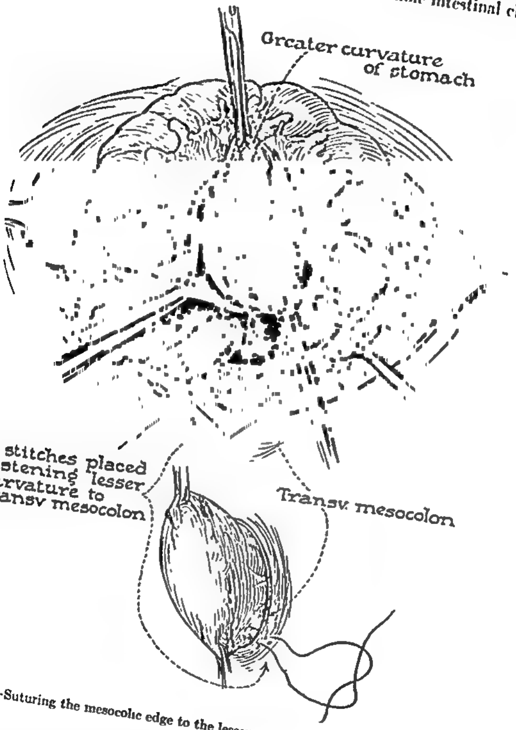


FIG. 338.—Suturing the mesocolic edge to the lesser curvature side of the stomach.

latter can be either Roosevelt, Linnartz, or single clamps. Linnartz and Roosevelt are three-bladed clamps with a fixed center blade. The Linnartz is the shorter of the two and is more flexible. The first loop of jejunum is now picked up and placed in the second blade of the intestinal clamp. Avoid clamping or including the mesentery of the bowel. The purpose of the clamps is to control the contents, and

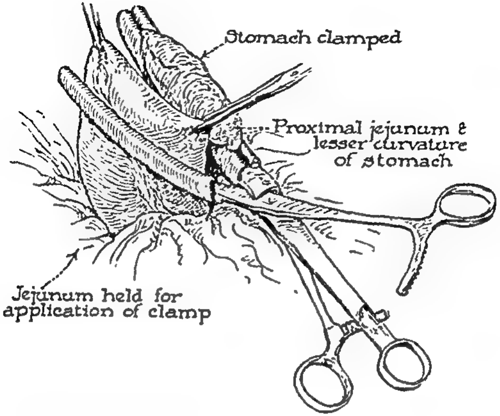


Fig. 339.—Stomach and jejunum in Linnartz clamps. The proximal end of the jejunum is approximated to the lesser curvature while the distal end is placed to the greater curvature.

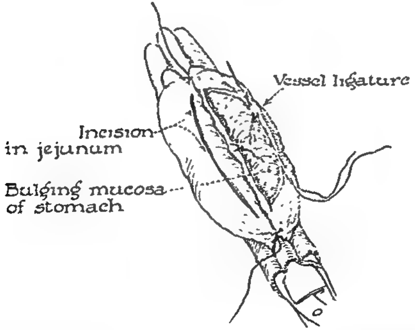


Fig. 340.—After the application of the Cushing suture, incisions are made in the stomach and jejunum. The jejunal opening is slightly shorter than that of the stomach. Illustrates method of ligating vessels in the gastric wall.

to facilitate suturing. Clamping too tightly should be avoided, as this may traumatize the mucus membrane. The proximal end of the jejunum is proximated to the lesser curvature (Fig. 339). The bowel is rotated so that its anterior surface is approximated to the stomach.

The Anastomosis.—The next step is to apply the first outer row of sutures. This is started with a mattress stitch and continued as Cushing stitches. The latter are placed ¼ inch apart on alternate sides of the stomach and intestine. They do not enter the lumen but are inserted just down to the mucus membrane. When about 3 inches of bowel have been sutured, a Lembert stitch is applied, locked, and the suture is saved for suturing the anterior outer row.

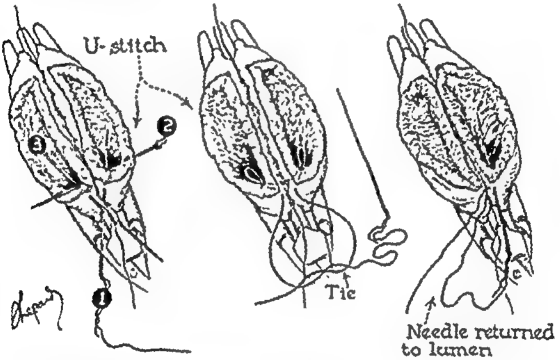


FIG. 341 FIG. 342 FIG. 343

FIG. 341.—Method of inserting the U-stitch: (1) starts from without in; (2) through the septum; and (3) out again.

FIG. 342.—U-stitch completed and ready to be tied

FIG. 343.—After the U-stitch has been tied, the needle re-enters the lumen.

The incisions into the stomach and jejunum are now made. Make the stomach incision at least ¼ inch from the end, ¼ inch from the beginning, and ¼ inch parallel with the row of Cushing sutures. The incision is first outlined on the serosa, muscularis, and submucosa. By allowing the mucosa to bulge, the blood vessels are exposed, and can be ligated by inserting fine catgut stitches close to the edge of the incision (Fig. 340).

Complete the opening by cutting the mucus membrane. If the mucus membrane is redundant, it may be removed. This makes suturing easier and prevents the formation of a large septum. A similar incision is made in the jejunum, except that it is made shorter than the stomach opening. If the stomach is dilated, or atonic, the jejunal incision should be made about ½ inch shorter. Outline the incision first on the serosa, then make a stab wound at the lower angle and gently express the content. The incision is completed with scissors. The opened stomach and intestine are now sponged out, being careful not to injure the mucosa.

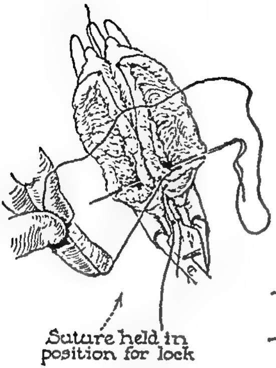


Fig. 344

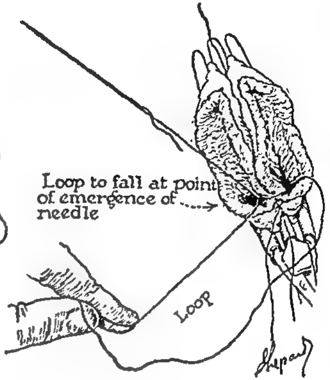


Fig. 345

Fig. 344.—Method of suturing the septum with a continuous lock-stitch.
Fig. 345.—The suture is held so that the loop will fall at the point of emergence of the needle

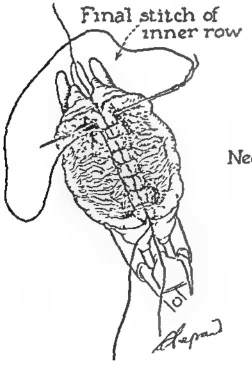


Fig. 346

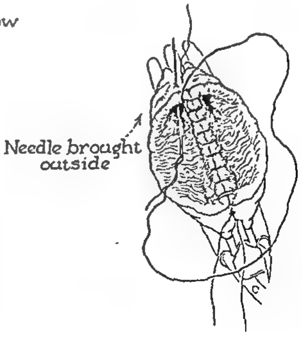


Fig. 347

Fig. 346.—Final stitch of the septum suture.
Fig 347.—Beginning to invert the corner.

The inner, hemostatic row of suture is now started. This is begun as a U-stitch. As illustrated in Figures 341 to 343, the needle passes from without-in, through the septum, then out again opposite to the point of entrance. The suture is tied, and a forceps left on the short end. This acts as a landmark when completing the anterior row of the Connell sutures. After tying the U-stitch, the needle re-enters the lumen, and the posterior edges (septum) are sewed with a simple over-and-over lock stitch. While suturing, keep the line taut with the left hand. The suture can be self-locked by throwing forward a loop of the suture material, thus; when the needle is pulled, the stitch will lock itself.

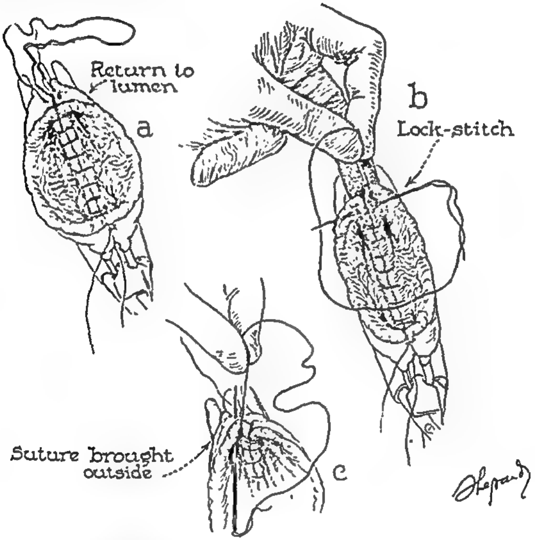


FIG. 348.—(a) Completing the inversion of the corner by re-entering the lumen; (b) after turning the corner, the angle is reinforced with a lock-stitch; (c) the needle is carried from within-out and the anterior edges are now inverted

After the entire septum has been sutured, the angle is inverted by continuing with the same suture. As illustrated in Figure 348, the corner is inverted by passing the needle out on one side and re-entering the other side. As a rule, one stitch is sufficient for turning the corner; however, if necessary, the same process is continued until the corner is satisfactorily inverted.

The same suture is now continued to sew the anterior edges with the Connell suture. This is applied as follows: The needle is passed from without-in, then out on the same side advancing about $\frac{1}{8}$ inch. The needle is then passed to the other side re-entering the lumen without advancing, then out again on the same side

nor too close to the edges. It has been found that when they are applied ¼ inch apart, and ¼ inch from the edges, inversion is effected without puckering and with the least possible danger of hemorrhage. The advancing is done from within-out, and never from without-in. Advancing from without-in causes puckering and eversion of the edges, and as a result hemorrhage and leakage may occur.

After finishing the inner row, the clamps are removed, and the operating field is cleansed. Gloves are either washed or changed. The suture line is inspected for bleeding points and puckering, and if puckering is present, mattress sutures are inserted to invert it.

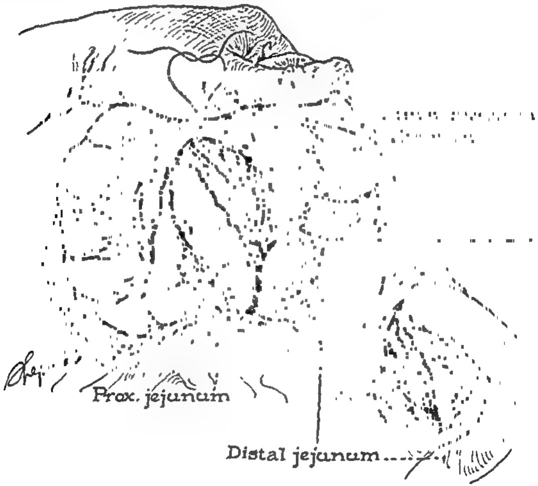


Fig. 352.—The remainder of the mesocolic edge is sutured to the stomach

The original posterior row of Cushing stitches is now continued on the anterior surface. Figure 351 illustrates the method of holding the anastomosis while applying the Cushing suture row. When completed, the suture is tied to the original short end of the posterior outer row. The angles are reinforced with one or two interrupted stitches.

The mesocolic edge is now sutured to the stomach side of the anastomosis. It is important that the mesocolon is adequately sutured to prevent herniation of the anastomosis into the lesser omental bursa. Furthermore, the anastomosis will drain better, and the jejunum will not be under tension.

ANTERIOR GASTROJEJUNOSTOMY

When performing an anterior gastrojejunostomy, a loop of jejunum well away from the ligament of Treitz is brought anterior to the transverse colon, and anastomosed to the anterior surface of the stomach. The anastomosis is made in an isoperistaltic manner, i. e., the proximal end of the jejunum is approximated to the cardiac end of the stomach, and the distal end to the pyloric end. The incision on the stomach is made parallel with the greater curvature, with the distal end made a little higher than the proximal.

The technical steps in the anastomosis are the same as described for the posterior operation. To prevent stagnation in the distal loop of the jejunum an antero-enterostomy is done, 4 to 6 inches below the gastric anastomosis.

(Questionnaire on page 625.)

There are numerous methods of resecting the stomach; however, the underlying principles of all of these are based upon the Billroth I and Billroth II methods (Fig. 353). It makes little difference what type of resection and anastomosis is made, provided the method is sufficiently radical to permit a wide resection. For carcinoma of the stomach, the operation to be effective must remove the following parts: (1) the diseased portion of the stomach, including all of the lesser curvature and three-fifths of the greater curvature; (2) at least 1 inch of the duodenum,

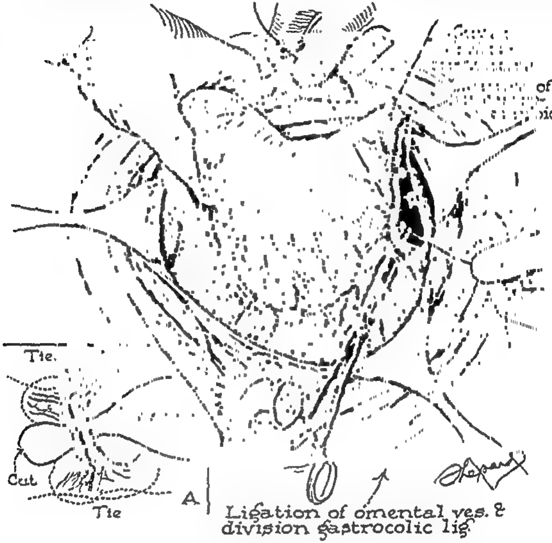


Fig. 356.—Separating the stomach from the colon. Insert illustrates the scheme in the division of the omentum.

as carcinoma may invade its wall; (3) the lesser and greater omentum and the gastrocolic ligament with lymphatic glands within them; and (4) lymph glands in relation to the posterior surface of the duodenum and pancreas. When the operation is done for gastric ulcer, it should be sufficiently radical to decrease gastric acidity by removing enough secretory mucosa. This requires a removal of all of the lesser curvature and three-fifths of the greater curvature; if less amount than this is resected, the ulcer may recur.

The classical Billroth I operation consists of resecting a portion of the stomach and anastomosing its stump to the end of the duodenum. In reality, the operation is a pylorectomy, because only a small portion of the stomach can be excised, if

it is desirable to avoid tension on the suture line. For this reason, the operation is not applicable in cancer of the stomach, or in large, adherent, and eroding ulcers which do not permit duodenal mobilization. Another objection to the method is anastomosing the thick gastric wall to the duodenum without causing obstruction.

In the Haberer-Finney modification the end of the duodenum is inverted and its side is then anastomosed to the end of the stomach. This method avoids the danger

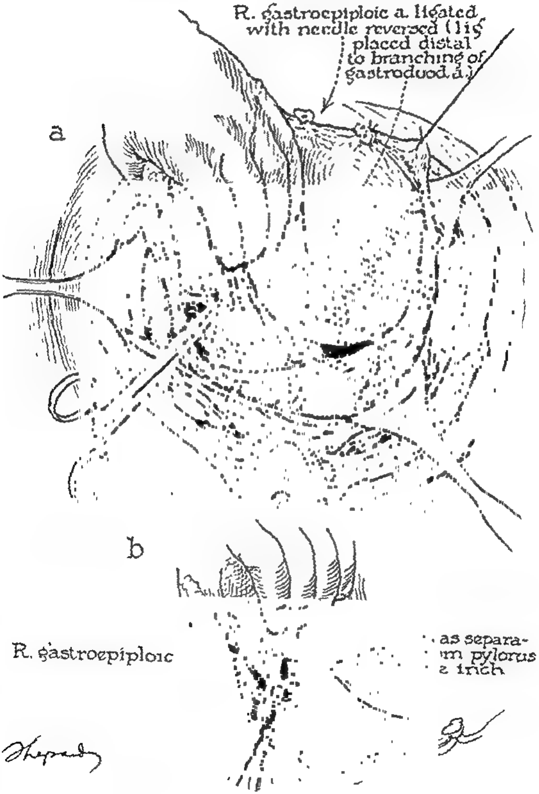


FIG. 357.—Ligation of the right gastric epiploic artery, and separation of the pancreas from the posterior surface of the duodenum.

from obstruction, but the tension on the suture line is increased unless the duodenum is mobilized. Horsley's modification of the Billroth I consists of increasing the circumference of the duodenum by making an extra incision, about 1½ inches, along the anterior wall of the duodenum. It is then sutured to the end of the stomach close to the lesser curvature, and, if end-to-end union is impossible, because of a wide stomach opening, the lower portion of the stomach is inverted separately.

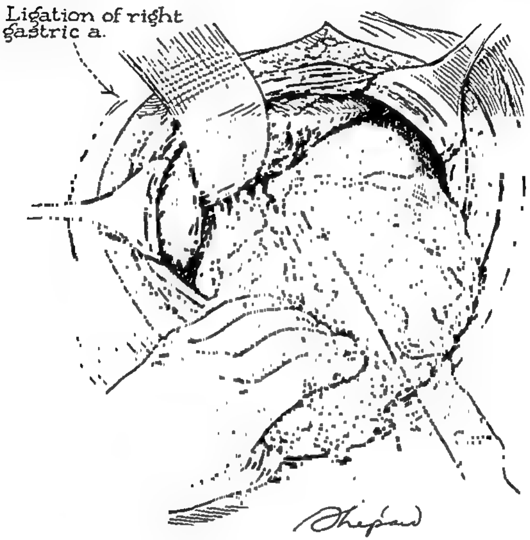


FIG. 358.—Ligation of the right gastric artery.

The classical Billroth II method of resection consists of closing both the duodenal and stomach ends, and performing a posterior gastrojejunostomy. The operation consumes a great deal of time and the posterior anastomosis may not be feasible after a wide resection of the stomach. It is of value as a two-step method when it is inadvisable to subject a patient to a one-stage radical resection. The Billbroth II operation has been generally discarded in favor of the Polya-Balfour or the Hofmeister-Finsterer method. In the original Polya modification the duodenal stump is inverted and the gastric opening is united to the first loop of jejunum through a rent in the mesocolon. The proximal end of the jejunum is placed at the lesser curvature and its distal end at the greater curvature. Balfour carries the jejunum anterior to the transverse colon and greater omentum and attaches the proximal end

of the loop to the lesser curvature and the distal end to the greater curvature. To avoid obstruction and interference in the circulation of the bowel, the posterior loop should be made rather long. If the posterior loop is too long Balfour advises an entero-enterostomy. Moynihan makes the anastomosis with the proximal end of the jejunum attached to the greater curvature and the distal end at the lesser curvature, hence avoiding the danger from vascular interference and obstruction which is apt to occur in the Balfour method. In the Hofmeister-Finsterer modi-

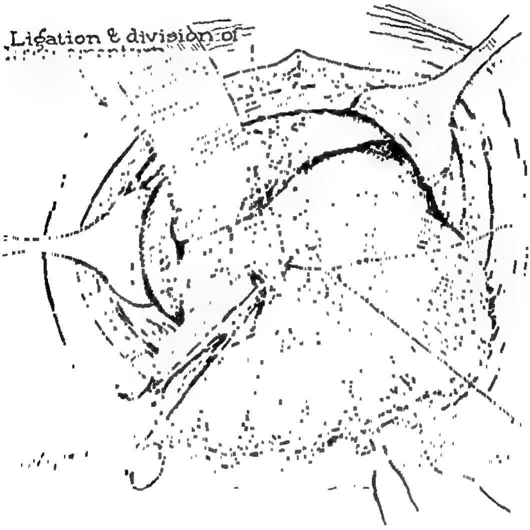


FIG. 359.—Separation of the lesser omentum.

fication only the lower portion of the gastric opening is united to the jejunum, the remaining part is *inverted*. The anastomosis can be made posterior to the transverse colon. The objection to this method is that leakage may occur at the convergence of the three suture lines. The Mikulicz modification consists of closing the gastric and duodenal openings and *uniting* the jejunum (through mesocolon) to the lower part of the stomach.

OPERATIVE TECHNIQUE

POLYA-MOYNIHAN MODIFICATION OF BILLROTH II

Exploration for Metastases.—Through a paramedian or Sloan's incision, the abdomen is opened and the stomach is examined to determine the *nature*, location,

and extent of the lesion. Should the lesion prove to be cancerous, the surgeon should, in a routine manner, seek for metastases in other organs. Since the spleen receives afferent lymphatic fibers from the left gastric epiploic nodes, it will be the first organ to become invaded when the cancer is primary in the fundus of the stomach. Metastastic growths in the spleen may also be present when the primary growth has started elsewhere in the stomach; however, splenic implantation in this instance is evidence of generalized metastases. If examination reveals that the

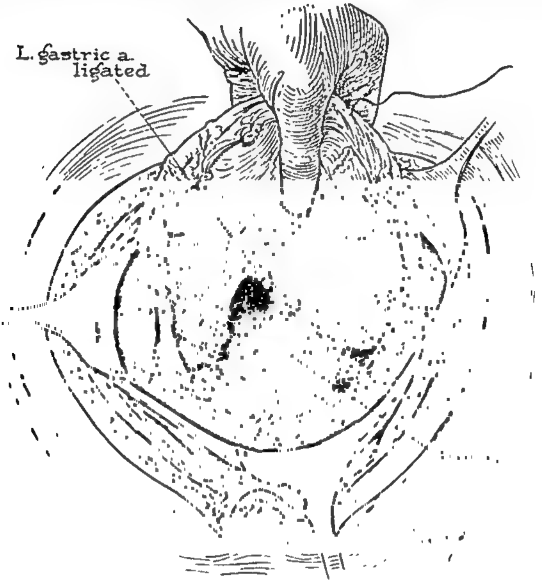


FIG 360—Ligation of the left gastric artery.

spleen is the only organ invaded with metastases, the operator must decide whether a palliative procedure, such as gastrojejunostomy or gastrostomy should be done as gastric resection is contraindicated.

If the spleen is free from metastasis, the colon is examined next. If the colon is involved, and no metastases are found in the liver, the probabilities are that the cancer had its inception at the greater curvature. Again, partial gastrectomy is contraindicated, and a palliative operation is done to relieve the patient from obstructive symptoms; if the mass is located close to the pyloric sphincter, a posterior gastrojejunostomy is made, and if it is located high up in the fundus, a gastrostomy is made for the purpose of introducing nourishment.

Involvement of the liver without evidence of metastases elsewhere, indicates that the carcinoma had its inception at the lesser curvature side of the pyloric end of the stomach. Sooner or later, tumor masses located in this area produce pyloric obstruction, hence if radical resection is contraindicated, the operation of choice is a posterior gastrojejunostomy to relieve the patient of obstructive symptoms. If examination reveals that none of the above organs and the rest of the abdominal and pelvic viscera are free from metastases, a partial *gastrectomy is indicated.*

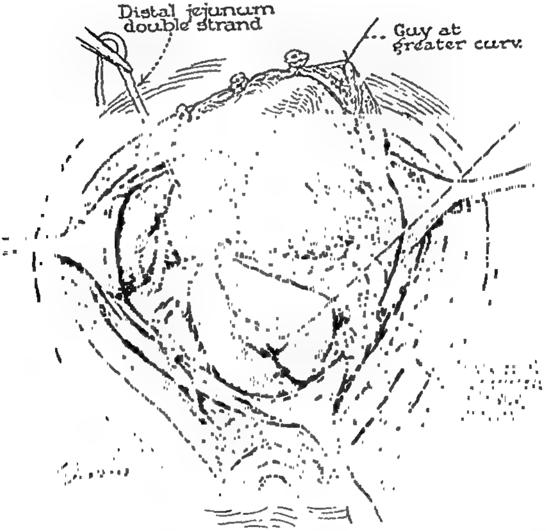


FIG. 361.—Identifying the loop of jejunum to be used for the anastomosis.

Enlarged lymph nodes in the lesser omentum or in the gastrocolic ligament are not contraindications to a radical resection, as the enlargements may be due to inflammatory reaction.

Mobilization of the Stomach.—After it has been decided that a partial gastrectomy is indicated, the first step is to determine the amount of stomach to resect. Regardless of the amount of stomach to be removed, the line of section of the duodenal side is practically the same, *i.e.,* at least 1 inch of the duodenum is removed. Unless otherwise indicated, the entire lesser curvature and three-fifths of the greater curvature should be excised. Place guy stitches at the limits of resection on the lesser and greater curvatures (Fig. 354). If possible these should include any large vessel that may be present. If difficulty is encountered in keeping the

Fig. 362.—A pad is placed between the curvatures, another in Morrison's pouch, and another over the transverse colon.

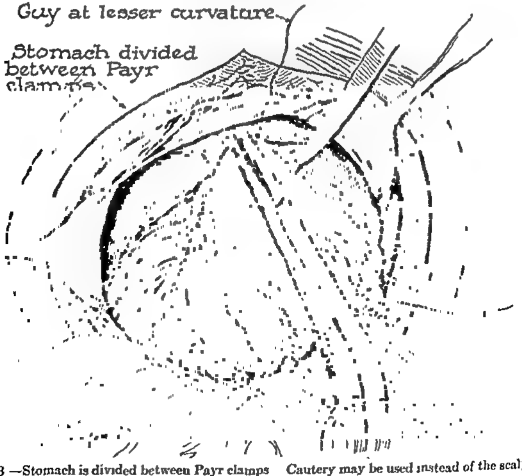


Fig. 363 —Stomach is divided between Payr clamps Cautery may be used instead of the scalpel if the excision is done for malignancy.

stomach within the abdominal incision, a laparotomy pack may be applied in the space left vacant after the fundus is delivered into the abdominal incision (Fig. 355).

Having placed guy sutures on the lesser and greater curvatures, the mobilization of the stomach is now begun by freeing it from its omental attachments. In order to avoid injuring the mid-colic vessels many surgeons begin with the division of the lesser omentum. We have found that it is easier and more practical to divide the gastrocolic omentum first. If the latter is firmly adherent to the transverse mesocolon, a small opening is made in the gastrocolic ligament and by finger dissection it is carefully separated.

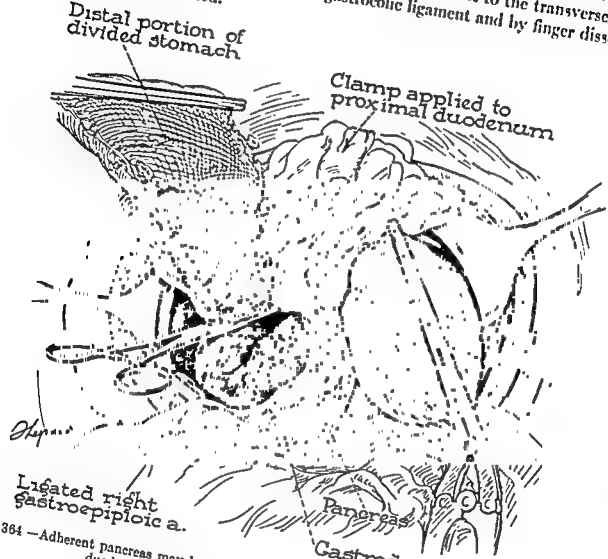


FIG 364 —Adherent pancreas may be freed at this stage, and Carmalt forceps applied on the duodenum at least one inch beyond the pyloric sphincter.

If the left gastro-epiploic vessel has not been included in the guy stitch, it is now ligated. The ligation is done by applying the stitch around the vessel and including a portion of the stomach wall. Make a small opening in the gastrocolic omentum and insert a finger through the rent to determine if the mesocolon is attached. If it is found adherent, it is separated gently, being careful to avoid injuring the middle colic artery. At times it may be necessary to remove avascular portions of the mesocolon that are attached to the gastrocolic ligament. The opening thus made in the mesocolon is closed with sutures, or the rent may be utilized later for bringing the jejunal loop through it, if a posterior anastomoses is decided upon.

The gastrocolic ligament is separated entirely from the stomach and the division is done between ligatures, as illustrated in Figure 356. If the lesion in the stomach is a malignant one, the omentum is separated as close as possible to the transverse colon in order to remove all lymph glands. In non-malignancy, the division can be

FIG. 365.—Division of the duodenum between two Carmalt forceps. Insert illustrates the Kerr-Parker inversion of the duodenal stump.

made close to the stomach. Gastrocolic omentum is divided as far as the duodenum, where a search is made for the right gastro-epiploic vessel. When found, it is ligated by inserting a curved needle (reversed) around it (Fig. 357, *a*). The artery should be ligated as it branches from the gastroduodenal artery. The latter is situated between the pancreas and the duodenum, thus to isolate the gastroduodenal artery

it is imperative to separate the pancreas from the duodenum (Fig. 357, *b*). This procedure causes a variable amount of oozing which can be easily controlled by hot application, or light ligatures. If the pancreas is markedly adherent to the duodenum the separation may be postponed until after the stomach has been divided, and turned toward the right, thus allowing more direct inspection of the posterior surface of the pyloroduodenal pancreatic region, and the pancreas can be dissected and mobilized under direct vision.

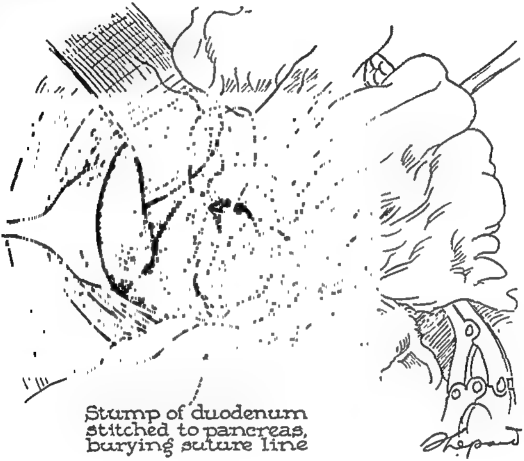


FIG. 366—Stitching the pancreas and omentum to the inverted duodenal stump.

After the right gastro-epiploic artery has been ligated, at least 1 inch of the duodenum is cleared of omental fat and detached from the pancreas. This is an important procedure since the end of the duodenum must be subsequently inverted. If the resection is done for a posterior duodenal ulcer one must be careful while separating the pancreas, as the ulcer may have eroded into it. In such contingency, the pancreas may be left attached to the duodenum and the division made proximal to the adherent ulcer. An ulcer which is permitted to remain is sidetracked from food and acid gastric juice and will be constantly bathed in alkaline duodenal fluid.

Having mobilized the greater curvature of the stomach and the first part of the duodenum, the right gastric artery is identified and ligated within the duodenohepatic ligament (Fig. 358). This procedure can be facilitated by introducing a finger underneath the pylorus and directing it cephalad toward the ligament where the vessel may be made prominent. Instead of using the finger, the author introduces a Cameron surgilite behind the pylorus and, by pushing it forward, the artery is transilluminated, and ligated under direct vision. The right gastric artery is im-

bedded in soft tissue, and when severed it will instantly retract, hence when ligating, the ligature must be securely tied. Should the ligature slip, the bleeding point may be located by placing the index finger into the foramen of Winslow, and the thumb anterior to the duodeno-hepatico ligament; then to alternately pressing and then releasing the hepatic artery, the bleeding point is determined, and the divided artery is religated.

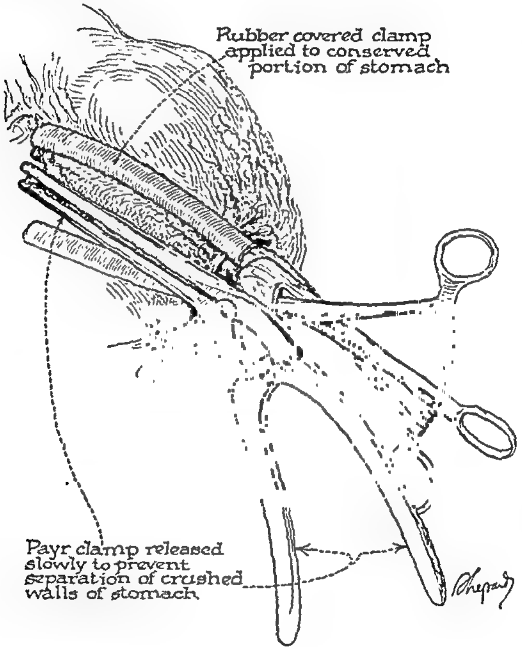


FIG. 367.—Linnartz clamps applied on the stomach.

Now the lesser omentum is divided between ligatures in the same manner as the separation of the gastrocolic ligament (Fig. 359). The division is carried as far as the guy suture at the upper limit of section on the lesser curvature. If the resection is done for malignancy, the separation is made close to the liver. At this stage of the operation, all of the arteries of the stomach, except the left gastric, have been ligated. The left gastric can now be easily ligated by lifting the greater curvature of the stomach to the right and ventrally, thus placing the vessel "on the stretch"

(Fig. 360). Lymphatic glands are stripped toward the stomach, and the bare vessel is ligated close to its origin. In cases of benign lesions, when a lesser amount of stomach is excised, the vessel may be ligated close to the lesser curvature, and if there are numerous large branches present, they are ligated individually.

Excision of the Stomach.—Before proceeding with the excision of the stomach, it is a good plan to locate the loop of jejunum which is to be used for the anastomosis. The greater omentum and the transverse colon are raised and the first loop of jejunum is identified. A loop of jejunum, about 10 to 18 inches from the duodenal-jejunal flexure, is carried *in front* of the transverse colon, and the portion to

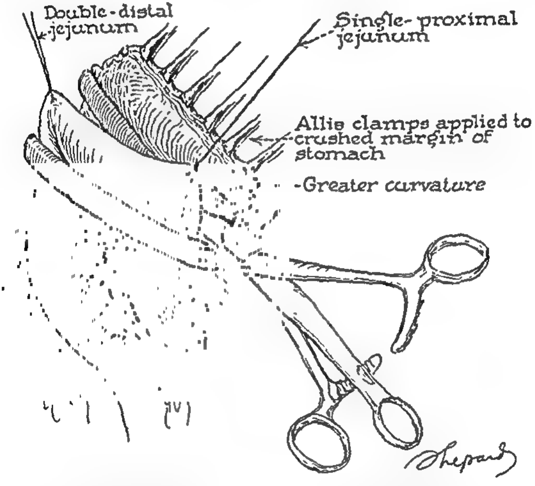


FIG 368 —Approximating the jejunum to the stomach; proximal end to the greater curvature and the distal end to the lesser curvature (Moynihan).

be employed for the anastomosis is marked off. This may be done by applying a single stitch at the proximal end of the loop and a double stitch at its distal end. Instead of applying stitches, the loop may be marked off by passing a single strand through an avascular area of the mesentery to indicate the proximal end, and a double strand to indicate the distal end (Fig. 361). The loop, which is then placed back into the abdomen, may be pulled out when needed. In this manner, when the loop is required for the anastomosis it can be withdrawn without the necessity of entering the abdomen with soiled gloves.

To prevent soiling, the abdomen is walled off by placing a laparotomy pad in Morrison's pouch, one over the liver, and another between the lesser and greater curvatures (Fig. 362). The stomach is now grasped with a Payr clamp on the line

between the two guy stitches on the lesser and greater curvatures. The purpose of the Payr clamp is to crush the edges of the stomach, to obliterate small blood vessels and capillaries, and to prevent escape of content. Another Payr clamp is applied distal to the first one, and the stomach is incised in between the two clamps (Fig. 363). The cut surface is cauterized with the actual cautery to avoid cancer cell transplantation, as well as for hemostatic purposes. The gastric stump held in the Payr clamp is covered with gauze. The excised portion is turned to the right

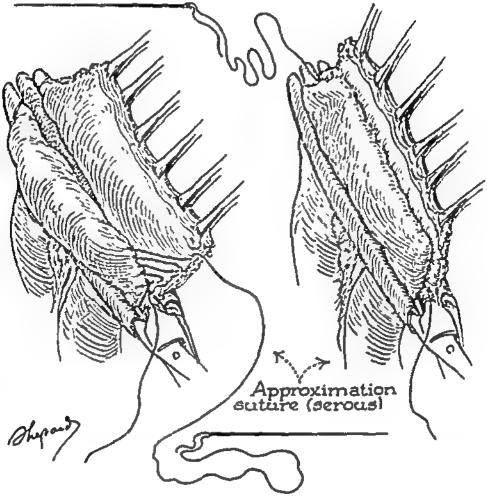


FIG. 369 FIG. 370

FIG. 369.—Partipilo method of building up the outer row of suture at the angle.

FIG. 370.—The first row of Cushing sutures has been applied. Note that at the lesser curvature the suture is carried downward in order to produce an outer row at the angle.

exposing the pancreas and the posterior surface of the pyloroduodenal region (Fig. 364). If the pancreas is adherent and has not been mobilized, it is freed with sharp dissection, being careful to avoid cutting its ducts. Injury to the pancreas can be prevented by leaving the serosa of the pylorus attached to the pancreas. Next, the right gastro-epiploic artery is ligated, if it has not already been secured. The duodenum is now grasped with Carmalt forceps and sectioned about 1 inch beyond the pyloric sphincter (Fig. 365). The cut surface of the duodenum is cauterized and the diseased stomach is discarded.

The duodenal stump is inverted by the Kerr-Parker method. If the duodenal stump is short, it is held open with Allis forceps and the closure is accomplished with a continuous row of Connell suture. A second row of suture is applied for

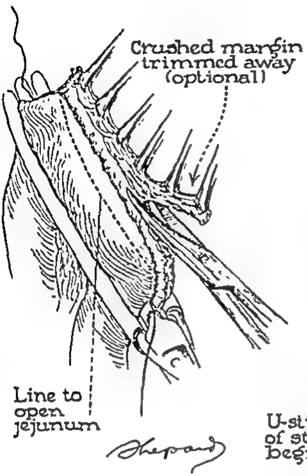


Fig 371 Fig. 372

Fig. 371.—The line of incision on the jejunum is shorter than the stomach opening. The crushed portion of the stomach may be trimmed away.

Fig. 372 —Beginning of the inner row of suture with a U-stitch.

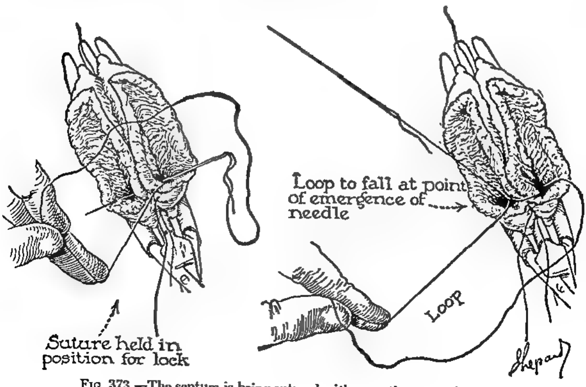


Fig. 373.—The septum is being sutured with a continuous lock-stitch.

reinforcement. The pancreas and the omentum are drawn over and fastened to the duodenal stump to cover any denuded area of the pancreas, and also to further obviate the possibility of leakage from the duodenum (Fig. 366).

Anastomosis.—The first stage of the operation which is now completed, comprises undoubtedly the most difficult part of the entire procedure. The routine

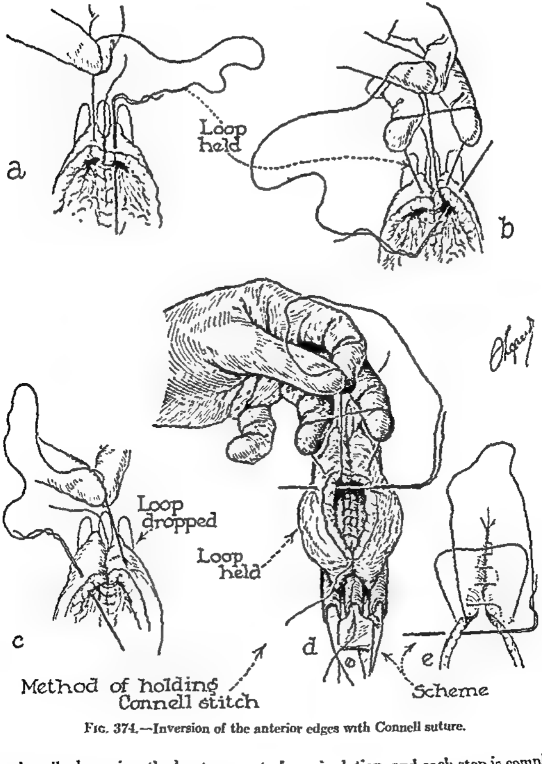


Fig. 374.—Inversion of the anterior edges with Connell suture.

just described requires the least amount of manipulation, and each step is complete in itself so that it is never necessary to retrace or complete any unfinished step.

The second stage of the operation consists of anastomosing the end of the stomach to the side of the jejunum. Place a rubber-covered intestinal clamp on the stomach below the Payr clamp (Fig. 367). Bring the loop of jejunum out of the

abdomen by pulling on the ligatures. It will be recalled that a single ligature through the mesentery designates the proximal end, and a double ligature the distal end. As previously stated, the jejunum is carried over the transverse colon and the greater omentum. Place the jejunum into the other blade of the intestinal clamp (Fig. 368). The proximal end of the jejunum is proximated to the greater curvature, and the distal to the lesser curvature (Moynihan). Make certain that there are no kinks or twists in the distal segment of the bowel. Balfour places the proximal end at the lesser curvature, and the distal end at the greater curvature. We have found that when the approximation of the jejunum is made in this manner, the distal loop necessarily becomes kinked, thus predisposing to obstruction.

FIG. 375.—The operation is completed by attaching the lesser omentum to the anastomosis

The first row of suture can be applied with greater ease if the Payr clamp is removed and the agglutinated septum is held together with Allis forceps. The suture is begun at the greater curvature side with either a Lembert or a mattress stitch. Figure 369 illustrates the author's method of "building up" an outer serosal layer at the angle. Notice that the initial stitch is taken about ¾ of an inch below the cut margin, then continued as Lembert stitches on the stomach side only until the edge is reached, when the outer row is completed with Cushing stitches (Fig. 370). The angle at the lesser curvature is similarly treated. The opening in the jejunum is made, and should be slightly shorter than the stomach opening. If it is desirable, the crushed margins of the stomach may be excised with scissors (Fig. 371). However, it is not necessary to do so as the inner row of suture, being

hemostatic, will produce sufficient pressure to cause the crushed margins to slough off. If the crushed margin is excised, there will be large bleeding points which must be clamped and ligated. After the Allis clamps are removed, the jejunum and the stomach stump are cleansed. The inner row of suture is started at the greater curvature with a U-stitch and tied (Fig. 372). Then, re-entering the lumen, the septum is sutured with a simple over-over continuous lock stitch. When the corner is reached, it is turned and the suture continued as a Connell, inverting the anterior edges. When the short end of the original inner row is reached, the suture is tied and the ends are cut.

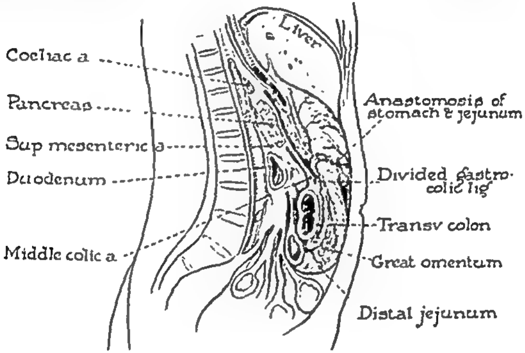


FIG 376.—Schematic drawing illustrating the relationship of the anastomosis with the transverse colon.

At this time, the operative field is cleaned, the flexible intestinal clamps removed, and the gloves changed or cleansed. The original posterior outer row is now continued on the anterior. When finished, interrupted sutures are applied to reinforce the angles. The anastomosis is inspected for points of eversions and hemorrhages. If the gastrocolic omentum is long enough, it is sutured to the lesser curvature, if not it is attached to the greater curvature (Fig. 375). After final inspection is made, the abdomen is closed in the usual fashion.

QUESTIONNAIRE ON PAGE 625

CHAPTER 53

GASTRIC SURGERY—RADICAL GASTRECTOMIES

By Robert L. Schmitz

GENERAL CONSIDERATIONS OF CANCER OF THE STOMACH

The curability of cancer of the stomach by surgical removal has been well established; yet, the over-all prognosis is poor. Although the main problem is with earlier diagnosis, inadequate treatment accounts for a percentage of failures. All but the hopelessly advanced cases should be explored to determine what can be accomplished.

Curative surgery implies the removal of an adequate portion of the stomach and its regional lymph node deposits in continuity (Figs. 377–378). On occasion, portions of neighboring organs may be included in the excision in an attempt to encompass direct extension of the gastric lesion.

Total gastrectomy has been recommended as the routine procedure for gastric cancer but clinical evidence is lacking to prove that it is any more curative than a radical subtotal gastrectomy to justify the greater morbidity and mortality attached to it. The greater and lesser omenta and regional lymph nodes can be removed very adequately with a subtotal resection and any portion of the stomach saved permits a more secure anastomosis and a more comfortable patient from the standpoint of eating, digestion and nutrition. However, the tendency of gastric cancer to spread submucosally makes it imperative to resect 5 cms. or more beyond the gross edges of the neoplasm. Neither the duodenum nor the esophagus are barriers to this spread, therefore adequate portions of these structures must be included in the excision when the neoplasm extends into their neighborhoods.

In the majority of cases, the lesion is in the distal two-thirds of the stomach and enough of the esophago-gastric junction is free of disease beyond the tumor to permit the surgeon to spare the cardia. As a rule, in this situation 80 to 90 per cent of the stomach is sacrificed. In the less common lesion confined to the upper third of the stomach, the antrum can often be spared. Intestinal continuity is re-established by esophago-antrostomy, below or above the diaphragm as the situation demands. When the cancer lies close to both the cardia and pylorus or when it is diffuse, as in linitus plastica or lymphosarcoma, the surgeon has no choice but to perform a total gastrectomy.

When the disease is incurable because of irremovable extension or metastasis, partial gastrectomy is still indicated, if technically possible, since one or two years of comfortable extension of life is not unusual following it. If palliative resection is technically impossible, lesser procedures, such as gastroenterostomy, gastrostomy, or jejunostomy may be indicated.

Preoperative Preparation.—The general status of the patient is appraised, especially from the standpoint of concurrent or chronic disease and distant metastasis, *i.e.*, hepatic, pulmonary, skeletal. Anemia is common and is corrected with transfusions. Electrolyte imbalances especially of chlorides, sodium or potassium

may be present if the patient has been vomiting, and are balanced by parenteral replacement. The patient is often malnourished, therefore the serum proteins and A/G ratio are determined and any deficiency is replaced by whole blood plasma and parenteral amino acid administration. One thousand cubic centimeters of whole blood are cross matched and ready before surgery. If there has been obstruction, it is well to decompress the stomach by constant suction through a nasogastric tube for one or more days preoperatively.

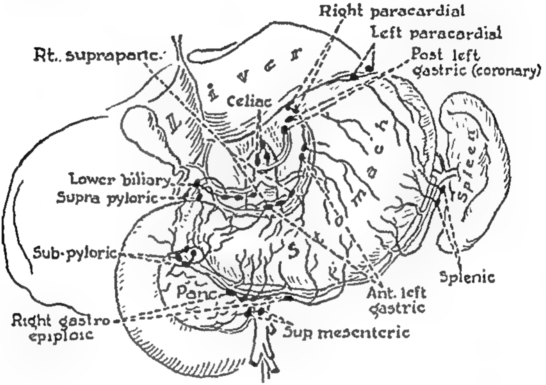


FIG. 377.—Lymph nodes of the stomach. Anterior view.

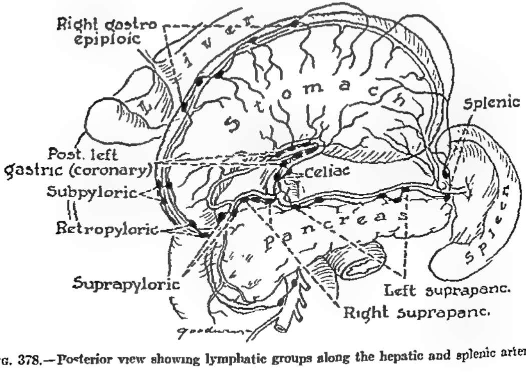


FIG. 378.—Posterior view showing lymphatic groups along the hepatic and splenic arteries.

Incision.—A left paramedian incision is made from the costal arch to the level of the navel for exploration (Fig. 379). After exploration, if only palliative short-circuiting operations are in order, the incision will suffice. If subtotal gastrectomy is the procedure of choice, the incision is extended downward to give additional exposure. If total gastrectomy is indicated, the incision is extended over the costal arch to the 5th or 6th interspace and the costal arch is divided without entering the pleural space. If further mobilization of the esophagus is necessary, the 5th or 6th interspace is incised; the pleural space is entered and the diaphragm is divided to the esophageal hiatus.

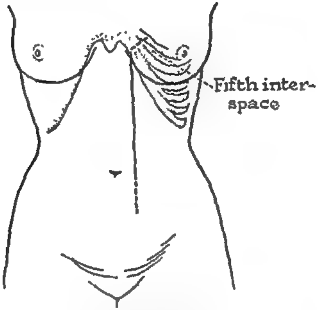


FIG 379.—Incision for radical gastrectomy. Solid line is the initial incision; dotted line indicates possible extension, if indicated.

Operative Appraisal.—As soon as the peritoneal cavity is opened, it is explored for secondary lesions from the mother growth; in particular, the cul-de-sac, ovaries, periaortic lymph nodes, small bowel mesentery, transverse mesocolon, liver and visceral and parietal peritoneum are inspected and palpated.

Next, the local extent of the primary lesion is measured. How much of the stomach seems involved grossly? How much of the lesser curvature? How close does it extend to the esophagus? To the pylorus? What regional lymph nodes seem to be involved? Is there extension to the transverse colon or mesocolon? To the pancreas? To some other organ?

On the basis of the findings, appropriate definitive surgery is executed.

RADICAL SUBTOTAL GASTRECTOMY

The greater omentum is delivered through the wound and retracted upward by the surgeon and his assistant. By sharp dissection immediately along the superior margin of the transverse colon the omentum is detached allowing the colon and mesocolon to drop away as the lesser omental bursa is entered (Fig. 380).

As the duodenum is approached the subpyloric nodes are carefully included in the dissection and then 3 to 5 cms. beyond the pylorus the line of excision is curved toward the duodenum, dividing and ligating the right gastro-epiploic vessels as

they are encountered (Fig. 380). As the pylorus and duodenum are reflected upward, any attachments to the pancreas are separated until the lesser curvature is reached. Anteriorly, along the superior edge of the gastro-duodenal junction the right gastric vessels are identified, divided and tied. This point of separation of the lesser omentum is extended upward to the undersurface of the left lobe of the liver and along it to the esophageal hiatus. Thus the hepato-gastric portion of the lesser omentum is separated from the hepato-duodenal portion (Fig. 381).

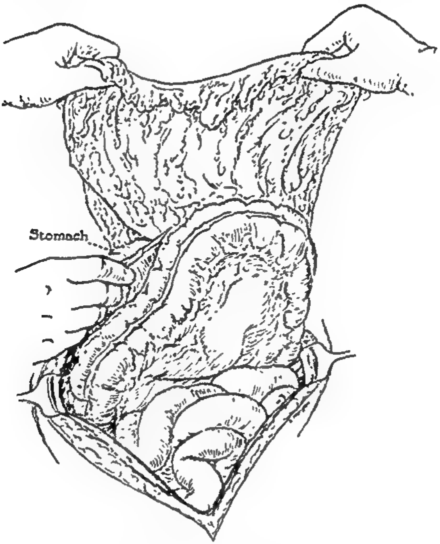
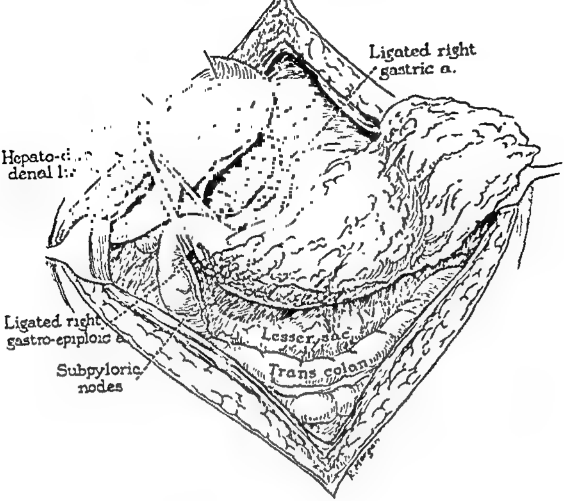


FIG. 380.—Radical gastrectomy. The omentum along the superior margin of the transverse colon is detached.

The entire greater curvature of the stomach with the attached greater omentum is reflected upward to expose the celiac axis, tail of the pancreas and splenic pedicle (Fig. 382). The left gastric vessels are isolated, divided and tied with silk close to the celiac axis. The attachments of the spleen to the posterior parietal peritoneum are separated by a sweep of the hand (*see* Splenectomy, Chap. 62). The spleen is rolled medially and the spleno-renal and splenocolic ligaments are divided as well as any remaining gastrocolic ligament. The spleen is lifted forward and a moist pack placed in the fossa. As much of the splenic pedicle as is feasible is separated from the superior margin of the tail of the pancreas. If nodal metastasis demands it, the tail of the pancreas can be included in the resection. The splenic



Greater omentum mobilized. Subpyloric lymph nodes re-
vessels divided and ligated.

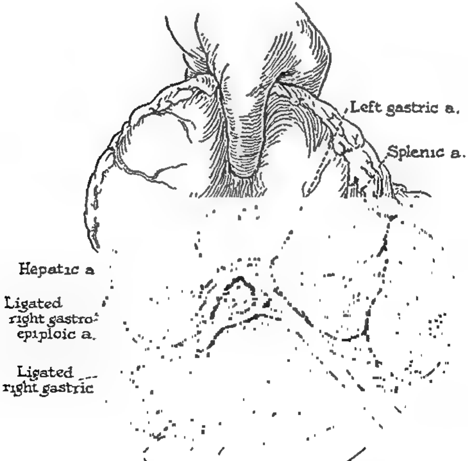


FIG. 382.—Radical gastrectomy. Stomach and greater omentum reflected upward; celiac axis, pancreas and splenic pedicle exposed. Left gastric artery being clamped before division.

artery is isolated and clamped with three Carmalt forceps; the spleen is gently massaged to empty it of blood; the splenic vein is then triply clamped. Division is made between the middle and distal clamps on the artery and vein and each is separately ligated with two suture ligatures of silk. The spleen is still attached to the stomach by the intact gastrosplenic ligament, with its lymph nodes. The avascular attachments of the cardia of the stomach to the diaphragm above the spleen are divided.

FIG. 383.—Radical gastrectomy. Spleen mobilized and pedicle transected and tissues turned upward. Gastro-jejunal anastomosis begun.

The duodenum is divided between Payr clamps at the site previously prepared. The entire distal stomach with attached omenta and spleen is reflected out of the upper angle of the incision onto the anterior thoracic wall where it is wrapped in a moist laparotomy pad (Fig. 383). The duodenal stump is closed in the usual manner.

A loop of jejunum about 35 cms. from the ligament of Treitz is brought up in front of the transverse colon (Fig. 383). The proximal portion of the loop is placed against the greater curvature and the distal portion against the lesser curvature across the posterior wall of the stomach just distal to the esophagogastric junction; about 10 to 20 per cent of the stomach is saved.

No clamp is placed on the stomach, a rubber shod clamp may be placed across both limbs of the jejunum well away from the site chosen for the anastomosis.

Individual vertical mattress sutures of 000 silk are placed through the seromuscular layers of the jejunum and posterior wall of the stomach and are tied (Fig. 383). The jejunum and posterior gastric wall are incised along this suture row and any leakage is aspirated or sponged away (Fig. 384*A*). A suture of 000 chromic catgut

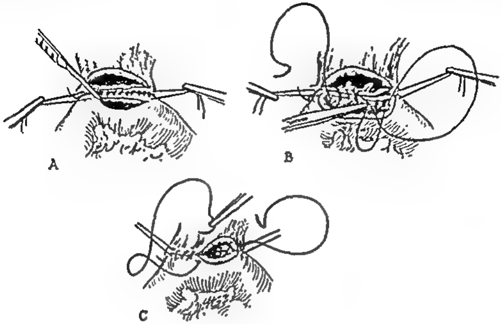


Fig. 384.—Radical gastrectomy. Anastomosis of the jejunum and gastric stump.

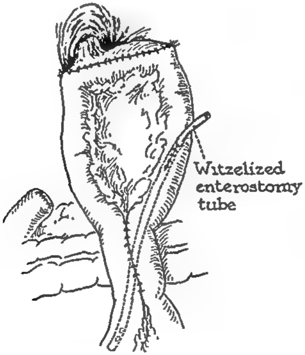


Fig. 385.—Radical gastrectomy. Anastomosis completed; entero-enterostomy completed; enterostomy tube in place.

with a curved needle swedged on either end is placed through all layers of the posterior edge of the anastomosis at its mid-point and tied in a single granny. One strand of the suture is sewn toward one angle and the other strand of the suture toward the other angle in a simple running stitch (Fig. 384, *b*). The anterior wall of the stomach is now transected detaching the entire specimen. The two catgut

strands are continued around either angle and along the anterior edge of the stomach and jejunum in the Connell manner until they meet at the midpoint where they are tied (Fig. 384, *c*). An anterior row of interrupted mattress sutures of silk completes the union (Fig. 385).

About 20 to 25 cms. below this anastomosis, just beyond the dependent level of the transverse colon, an entero-enterostomy 6 cms. in length is made between the afferent and efferent limbs of the jejunum, using an outer layer of silk and an inner layer of chromic catgut in the manner described above (Fig. 385).

Rather than a naso-gastric tube threaded through the pharynx, esophagus and the various stomata to be left for eight to ten days, a sterile Levine tube is placed into the afferent loop of the jejunum according to the Witzel technic (see Chapter 56) and the tip passed downward, through the entero-enterostomy into the distal jejunum, so that it can be left for feeding purposes for any length of time, according to the progress of the patient (Fig. 385). The tube is brought to the outside through a stab wound to the left of the incision and fixed to the skin with a silk stitch.

After the toilet of the peritoneum is completed, a Penrose cigarette drain is placed down to the duodenal stump and another along the gastro-jejunostomy into the splenic fossa. They are led out through the stab wounds at appropriate sites and the wound is closed.

TOTAL GASTRECTOMY

The operation proceeds as for subtotal gastrectomy. After the spleen has been mobilized, the stomach is allowed to fall back in the peritoneal cavity. The left lobe of the liver is retracted downward and the triangular ligament is divided. The lobe can now be folded under to the right from over the esophagus; and held away with a pack and retractor (Fig. 386, *A*). The diaphragmatic peritoneal reflection onto the esophagus is mobilized by sharp and blunt dissection (Fig. 386, *B*). Division of the vagus nerves will allow the esophagus to be drawn downward below the diaphragm for another 4 to 8 cms.

The duodenum is divided and the stomach, omenta and spleen are reflected upward through the wound onto the thoracic wall. The duodenal stump is closed

Occasionally, the Kocher maneuver may mobilize the duodenum enough to allow an end-to-end esophagoduodenostomy without undue tension but usually an esophago-jejunostomy is done to re-establish gastro-intestinal continuity.

As in the radical subtotal gastrectomy, a loop of jejunum about 35 cms. from the ligament of Treitz is brought up in front of the transverse colon (Fig. 387). An anastomosis is made between jejunum and esophagus using the same technique as is used for the high cardiac gastro-jejunostomy. After completion of the union, additional interrupted silk sutures are placed between the jejunal loop and the diaphragm on either side of the esophagus to take tension off the anastomosis (Fig. 388). The diaphragmatic peritoneal reflection dissected off the esophagus earlier may be tacked down over the anastomosis to help support it and seal it. The esophagojejunostomy may also be made in the Roux-Y manner (Fig. 388).

Again, an entero-enterostomy is done just below the dependent level of the transverse colon and a Levine tube is Witzelized into the afferent loop and passed through the entero-enterostomy into the distal jejunum and brought to the outside through a stab wound.

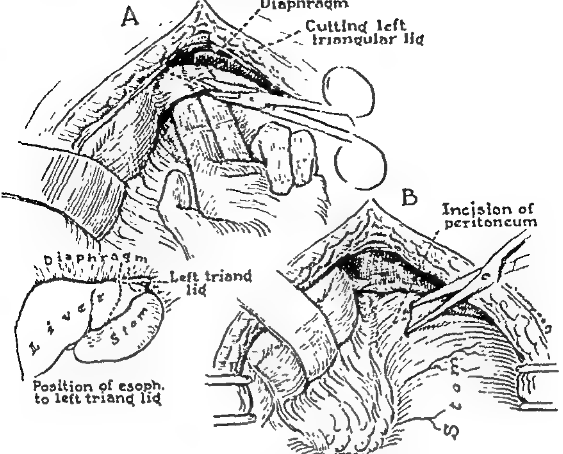


FIG. 386.—Total gastrectomy. A, Incision of the left triangular ligament; B, peritoneum over the esophagus is reflected on the diaphragm.

FIG. 387.—Total gastrectomy. Details of anastomosis.

MULTIPLE ORGAN RESECTION

If carcinoma of the stomach is localized and resectable except for extension into a neighboring organ, this organ, or an appropriate part of it, should be included in the excision in continuity, if possible. The organ is mobilized and included at the appropriate time and place during the gastrectomy. For example, the transverse colon and mesocolon can be included near the start when the greater omentum and greater curvature are being mobilized. The body and tail of the pancreas can be included at the time the splenic pedicle is being mobilized. All or part of the left lobe of the liver can be included toward the end of the operation just before or after the esophago-jejunostomy.

SUBSTITUTE STOMACHS

Two unpleasant sequellae to extensive gastrectomy are: lack of gastric reservoir and reflux esophagitis. To counteract them it has been suggested that various portions of the gastro-intestinal tract be interposed between esophagus and duodenum or jejunum, *e.g.*, a length of jejunum (Longmire), a pouch fashioned from a jejunal loop (Hunt), the transverse colon (State, *et al.*) and the ileo-cecal segment (Lee and Hunnicut). The results so far do not justify the addition of these long procedures to the end of an extensive operation which carries enough risk of its own.

POSTOPERATIVE MANAGEMENT

After gastrectomy the patient is given nothing by mouth until he has evidence of normal peristalsis, *viz.*, bowel sounds and flatus. Suction is applied to the enterostomy or naso-gastric tube during this interval. Parenteral electrolyte solutions are used to replace gastro-intestinal aspiration and to supply fluid for hydration and urine exeretion. When peristalsis has returned (normally about the 4th or 5th postoperative day) feedings of liquid formula are begun through the enterostomy or naso-gastric tube and the patient is allowed water by mouth in graduated amounts: 30 cc. per hour for the first 24 hours; 60 cc. the next 24 hours and 90 cc. the next 24 hours. When the patient can tolerate 90 cc. of water per hour, small feedings of soft foods are given at frequent intervals and gradually concentrated into six or eight feedings a day. The schedule of six or eight feedings is continued for three or four months after the operation and even thereafter if the patient finds he cannot get enough nourishment comfortably on less.

If at any time during the initiation of this program there is evidence of leakage at the anastomosis (left upper abdominal or lower thoracic pain, left shoulder pain, rising fever, suggestive drainage from the wound) the oral intake is stopped but the tube feeding is continued unless ileus is present. As soon as the symptoms have subsided, oral feedings may be resumed.

As soon as the patient is taking an adequate diet by mouth without untoward reaction, the enterostomy tube can be withdrawn. He will not tolerate large amounts of liquid at one time for quite a while because he has no gastric reservoir. Digestion of protein, carbohydrate and fat will be impaired, especially fat, and it is wise to increase the amount of fat in the diet. His weight will drop off to a new plateau which is usually considerably under normal. The stools become much softer and more frequent and bouts of diarrhea are common, especially early in the postoperative course

Most of these patients soon develop a microcytic, hypochromic anemia because iron absorption is impaired. Castle's intrinsic factor is lost when the stomach is resected and without it vitamin B_{12} is not absorbed from the gastrointestinal tract. The liver stores of B_{12} are large and are used up very slowly, hence it is several years before macrocytosis appears but if the patient lives long enough he will develop the picture of pernicious anemia. To avoid these complications, the patient should receive vitamin B_{12}, intrinsic factor and finely pulverized iron.

Many of these patients have one or more unpleasant symptoms when they eat: upper abdominal pain and distention, nausea, weakness, sweating, palpitation, dizziness and occasionally prostration, syncope or vomiting. We speak of the "dumping syndrome." The symptoms may mitigate or disappear with time but not infrequently continue to bother the patient as long as he lives. A high fat diet in frequent small feedings helps to some degree but there is no satisfactory treatment.

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With silk, linen, or gastro-intestinal catgut, a through-and-through stitch is taken at the apex of the posterior edges. The short end is cut and the two margins are then sutured together with a continuous over-and-over lock stitch (Fig. 394). When the angle is reached it is inverted and the same suture is used as a Connell to infold the anterior margins. When the anterior apex is reached, the suture is tied and the ends cut. The suture line is inspected for puckering and bleeding.

Gloves are washed or changed, after which a second row of Cushing suture is applied for reinforcement. The separated lesser omentum is sutured back to the lesser curvature, and a final inspection of the suture line is made. The abdomen is closed without drainage, unless gastric contents have been spilled.

ULCERS ON THE GREATER CURVATURE

Ulcers on the greater curvature of the stomach are removed in the same manner as those on the lesser curvature. F. Gregory Connell in 1929 suggested fundusectomy as a new principle in the treatment of peptic ulcers. He suggests this operation for the correction of secretory but not for motor abnormalities, such as persistent hypersecretion. The steps of the operation are similar to the technique of a V-shape section of the lesser curvature. Naturally sufficient stomach must be removed in order to reduce the amount of acid secreting mucosa.

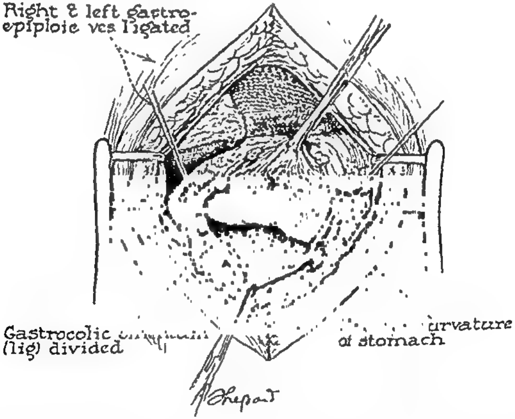

FIG. 395 —Ulcer on the greater curvature. Guy sutures have been applied and the gastro-colic omentum has been separated from the stomach.

ULCERS ON THE ANTERIOR SURFACE

Ulcers on the anterior surface of the stomach offer a somewhat different problem, although the principle of the operation is the same as when excising ulcers

on the curvatures. It is necessary in these cases to remove a diamond-shaped piece of stomach within which is located the ulcer (Fig. 396). The long axis of the diamond should be in the longitudinal axis of the stomach. Through an opening made in the gastrocolic omentum, intestinal clamps are applied to control the contents.

With the knife or cautery the incision in the stomach wall is made until the mucosa bulges. Large vessels are ligated close to the cut margin, and the excision is completed by cutting through the mucosa. The diseased part is discarded, and the opened stomach is cleansed. Beginning at the angle close to the lesser curvature,

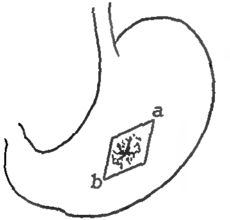

Fig 396

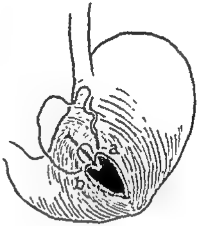

Fig. 397

Fig. 396.—Ulcer on the anterior surface of the stomach. Outline of the diamond-shaped piece of stomach to be excised.

Fig. 397.—Closure with Connell suture. Notice that the anastomotic line will be in the transverse diameter of the stomach.

the margins of the stomach are inverted with a Connell suture. The suture is applied first on one side then on the other, bringing the edges (a) and (b) together as illustrated in Figure 397. Notice that the suture line is in the transverse diameter of the stomach; thus the lumen is increased rather than decreased. After the inner row is completed, it is tied and ends are cut. A second outer row of Cushing stitches is applied and the operation is completed by suturing the rent in the gastrocolic omentum.

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CHAPTER 55

GASTRIC SURGERY—VAGUS NEURECTOMY VAGOTOMY

By A. V. Partipilo

DEFINITION

The word "vagotomy" is defined by Dorland as "the operation of cutting the vagus nerve." Therefore, the term should be reserved only for operations when the vagus nerves are sectioned but none is removed. When a part of both nerves is removed, the operative procedure is appropriately designated either as "vagus

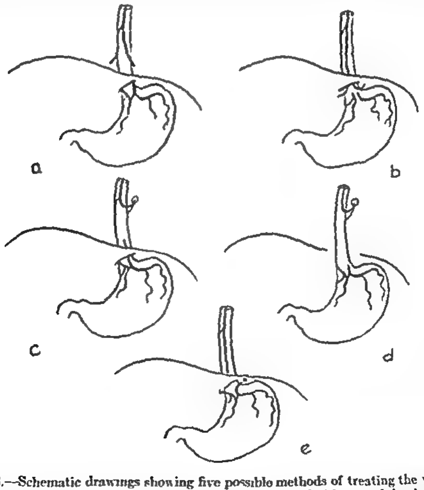

Fig. 398.—Schematic drawings showing five possible methods of treating the vagus nerves: (a) transthoracic vagotomy, nerves are sectioned but not excised; (b) transabdominal vagotomy, both nerves sectioned only; (c) transthoracic vagus neurectomy; (d) transdiaphragmatic vagus neurectomy; (e) transabdominal vagus neurectomy. (Modified after Moore.)

resection," or as "vagus neurectomy." To be complete, the definition should indicate whether the surgical approach is transthoracic or transabdominal, as well as whether the diaphragm is traversed. Hence, surgery of the vagus nerves may be one of five procedures (Fig. 398): (1) transabdominal vagotomy, when both nerves

are sectioned but none is removed; (2) transabdominal vagus resection or neurectomy, when both nerves are sectioned and segments are removed; (3) transthoracic vagotomy, when the vagus nerves are sectioned but none is removed; (4) transthoracic vagus resection or neurectomy, when segments of the nerves are removed; and (5) transdiaphragmatic vagal resection or neurectomy, when part of the vagus nerves is removed through a thoracic approach and the diaphragm is opened to remove the nerves down to their decussation on the stomach.

GENERAL CONSIDERATIONS

The addition of "vagus resection" to the surgeon's armamentarium in the treatment of peptic ulcer was presented as a practical procedure by Dragstedt and his co-workers in 1943. Since, then, it has been done by others and their reports indicate that the results obtained from this procedure are variable. That incomplete vagotomy is of no value in relieving peptic ulcer symptoms has been demonstrated by Weinstein, *et al.* In order to establish whether or not complete vagotomy has been accomplished, the Hollander insulin test must be done postoperatively. This test is based upon the physiological fact that hypoglycemia acts like a central nervous stimulus by causing secretory impulses to be transmitted by way of the vagus nerves to the gastric mucosa. In the normal adult, 20 units of "regular insulin" will cause the blood sugar to drop to 40 mgm. in thirty to sixty minutes with a resulting increase in the rate and acidity of the fasting gastric secretions. In the postvagotomy patient, there will be little or no free hydrochloric acid produced in response to the same amount of insulin. The blood sugar must be determined during the test and must be below 50 mgm. per 100 cc. of blood for the test to be valid. In most cases when vagus resection was done the secretory response to insulin-induced hypoglycemia remained abolished.

In order to perform a more complete resection of the vagus nerves, some surgeons prefer the transthoracic approach, because this route permits better visualization of the nerves. However, it has disadvantages common to any thoracotomy; namely, persistent painful scar, postoperative accumulations of fluid in the pleural cavity, atelectasis, pneumonia and bronchitis. The most serious objection to the transthoracic route is the inability to inspect and evaluate the existing pathology in the stomach and to carry out additional surgical procedures that might be indicated. In view of the malignant tendency of gastric ulcers, a surgeon would certainly hesitate to use the transthoracic approach.

The abdominal approach is preferred to the transthoracic route for the following reasons: (1) it permits direct exploration of the abdominal cavity; (2) it permits direct visualization and examination of the ulcer; (3) if the ulcer is suspected of being malignant, a gastric resection can be done; and (4) it permits some type of short-circuiting procedure if an obstructive ulcer is present. The objection that complete neurectomy cannot be performed through the abdominal route has been refuted by Bradley, *et al.* They have shown that in 92 per cent of cases, the nerves are sufficiently distinct so that their removal can be accomplished transabdominally. (See Chapter 49 for discussion of rationale of this procedure in the treatment of peptic ulcers).

SURGICAL ANATOMY

The surgical anatomy of vagal neurectomy is concerned with: (1) the relationship of the diaphragmatic portion of the esophagus, and (2) the distribution and relationship of the vagus nerves.

THE ESOPHAGUS

The thoracic esophagus lies posterior to the trachea as far as the fifth thoracic vertebra, where the trachea bifurcates. From this point downward, it is closely related to the posterior surface of the pericardium, and is separated from the vertebral column by the azygos vein, the thoracic duct, the intercostal arteries and, just before it pierces the diaphragm, the aorta. On the left side of the esophagus, in the upper part of the thorax, lie the left pleura and the left subclavian artery; in the region of the fifth thoracic vertebra, the aorta; and just before the esophagus pierces the diaphragm, the left pleura comes in contact with it. On the right side the esophagus comes in contact with the right pleura almost throughout its entire

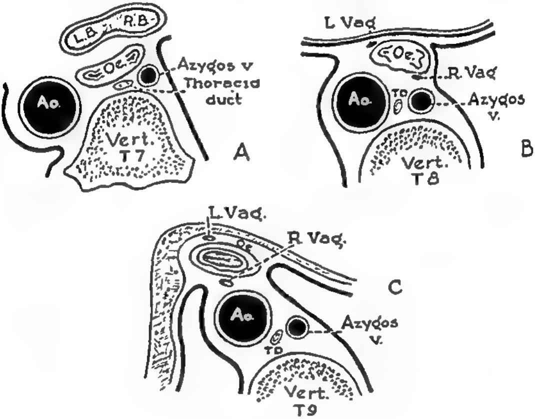

FIG. 399.—The relationships of the esophagus at various levels in the thoracic cage. (Redrawn after Cunningham.)

extent. Thus above the level of the arch of the aorta, the pleura, though not lying in immediate contact with the esophagus, is separated from it only by connective tissue. Below the arch of the aorta, the right pleura covers the right side of the esophagus, and very often a considerable portion of its posterior surface. On the left side, the left pleura comes in contact with the esophagus only for a short distance, just above the diaphragm, (Fig. 399). In this area the esophagus lies in close relation with the left pulmonary ligament. The latter is composed of two layers of pleura which come in close apposition at the root of the lung, and is prolonged downwards between the pericardium and the inferior surface of the lung and ending in a free border. The left pulmonary ligament is a landmark for locating the esophagus.

The diaphragmatic portion of the esophagus is about ½ inch in length and occupies the esophageal orifice of the diaphragm. As a rule the plane of the orifice

is oblique and to the left. The hiatus is embraced by two crura or pillars which consist of muscular fibers attaching the diaphragm to the vertebræ.

The abdominal part of the esophagus is about 1 inch in length. It lies in the esophageal groove of the left lobe of the liver underneath the left triangular ligament of the liver. This ligament is a continuation of the left layer of the falciform ligament. It is attached by one border to the superior surface of the left lobe near its posterior border, and by the other to the diaphragm for a distance of several inches. Its attachment to the diaphragm is to the left of the esophageal orifice and about ¾ inch anterior to the plane of this opening. Since this ligament is avascular, it may safely be severed in order to retract the left lobe of the liver when mobilizing the esophagus.

THE VAGUS NERVES

According to Cunningham, the vagi nerves, after reaching the root of the lungs, break up to form the posterior pulmonary plexus. From this plexus two nerves emerge to reach the esophagus and divide into small anastomosing branches which form the esophageal plexus. At the esophageal opening of the diaphragm two single nerves become separated from the plexus, and entering the abdomen, the left nerve is found in front of the esophagus, the right nerve behind it.

In an anatomic study of the vagus nerves in the lower part of the esophagus and upper part of the stomach Bradley, *et al.*, have shown that the right nerve was not always posterior nor the left nerve anterior to the esophagus as described by Cunningham. He has shown wide variations in the position, size and number of branches of the vagus nerves. Their studies were based upon observations made in more than 100 cases at necropsy.

In 92 cases the gastric nerves took origin from the esophageal plexus. In 85 of these cases, this occurred on the anterior wall of the esophagus behind the bifurcation of the trachea. In 7 cases this occurred at the level of the lower part of the esophagus. These 92 cases constituted three groups and the remaining 8 cases a miscellaneous group.

In the first group (64 cases) all the branches between the gastric nerves and from remote regions joined in common trunks, one on the right and one on the left side of the esophagus, somewhere between the esophageal hiatus and 6 cms. above the diaphragm (Fig. 400, *a*). In the second group (7 cases) there were many communicating branches between the main trunks that a plexus was formed over a major portion of the esophagus. The branches ultimately united at the esophageal hiatus (Fig. 400, *b*). The third group (21 cases) was characterized by long discrete trunks without any additions for a distance of 6 cms. above the diaphragm (Fig. 400, *c*). The fourth group was made up of the remaining 8 cases. In this group the nerves did not have a consistent or uniform course or pattern. In some instances well formed trunks would leave the diaphragm and course laterally in the thorax and lose their identity [illegible] branches of t[illegible] sides of the esophagus (Fig. 400, *e*).

As the trunks passed through the esophageal hiatus, the relative position of the vagus nerves to the esophagus was as follows: (1) left vagus; in 62 per cent it was found on the left anterior, 20 per cent left lateral, and 18 per cent left

posterior; (2) right vagus; 32 per cent right anterior, 24 per cent right lateral, and 44 per cent right posterior.

According to Bradley, the general distribution of the right and left gastric nerves below the diaphragm was as follows: *The right* gastric nerve coursed posteriorly and to the left. One of its branches followed the lesser curvature of the stomach as far as the *incisura*, and another large branch followed the left gastric

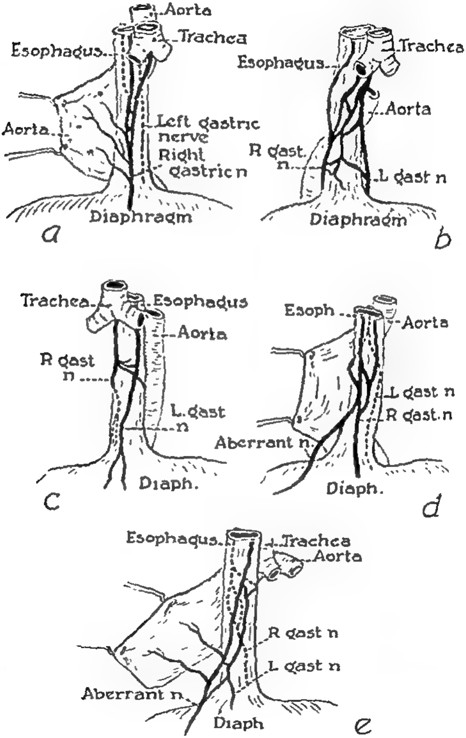

Fig. 400.—Variations of the vagus nerves: (*a*) group 1. Formation of the right gastric nerves from branches of the *pulmonary plexus behind the trachea and sympathetic* branches posteriorly; (*b*) group 2. Formations of gastric nerves from esophageal plexus; (*c*) group 3. Demonstrating the absence of communicating branches between the esophageal hiatus and [illegible] cm. above the diaphragm; (*d*) group 4. Right gastric nerve with aberrant nerve coursing laterally into thorax; (*e*) also group 4. Another example of distribution of nerves (Redrawn after Bradley *et al.*).

artery. The left gastric nerve almost immediately divided into numerous small branches which lost their identity in the serosa and musculature of the stomach.

The important observation brought out by Bradley, *et al.*, is that in more than 90 per cent of cases a transabdominal subdiaphragmatic surgical approach will allow as nearly a complete division of all gastric branches of the vagus nerves as will a transthoracic approach. In the remainder of cases, the pattern of the nerves is not uniform and some difficulty will be encountered in sectioning all the nerves.

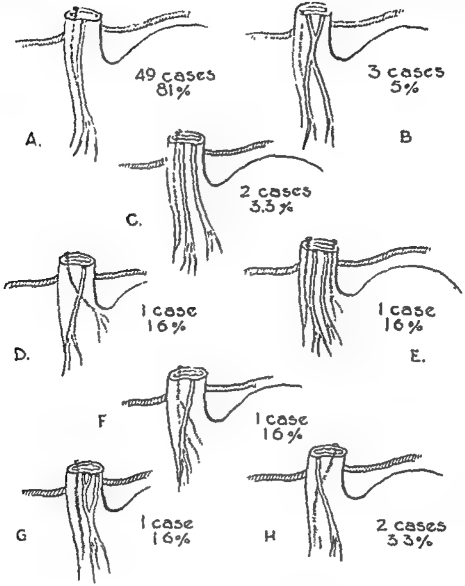

Fig. 401.—Sketches [illegible] along the lower portion of the esc[illegible] of the [illegible] Note that in 81 per cent

Figure 401 illustrates the distribution of the vagus nerve according to the studies of Dragstedt, *et al.* In 81 per cent of cases, the distribution of the vagus trunks is as illustrated in Figure 401, *a*. From their analysis of anatomical and operative findings, Dragstedt and his co-workers concluded that a complete vagotomy is just as successful by the transabdominal route as by exposure of the esophagus in the chest. They emphasize that in both the transthoracic and in the transabdominal operation, the vagus nerves are divided at a point $2\frac{1}{2}$ to 3 inches above the junction of the esophagus with the stomach. Hence, from a practical

standpoint, the operation is a supradiaphragmatic section of the nerves regardless of whether the approach is through the abdomen or through the chest wall.

TECHNIQUE—SUBDIAPHRAGMATIC VAGAL NEURECTOMY

Incision.—The incision for exposure of the abdominal esophagus may be either a midline, a high left paramedian, a left paramedian with an inverted T-leg, a left subcostal, or Sloan's transverse. The author has found Sloan's incision affords adequate exposure to the esophagus.

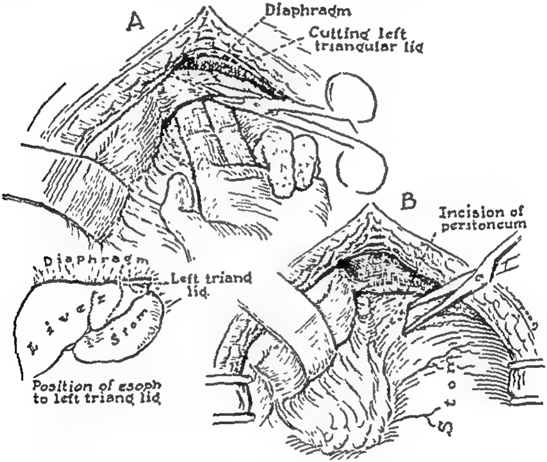

FIG 402 —(a) Incision of the left triangular ligament; (b) the peritoneum over the esophagus in incised at its reflection on the diaphragm.

Before proceeding with resection of the vagus nerves, the organs in the upper abdomen are examined. At this time the surgeon decides whether an additional operation on the stomach is indicated. Because of the dangers resulting from contamination of the subdiaphragmatic space during gastric operations, it is preferred that the vagus nerves be sectioned first. Furthermore, because of technical difficulties it may not be possible to resect the nerves and in that event the surgeon may want to perform a more radial gastric procedure than was originally contemplated.

Mobilization of the Esophagus.—The abdominal portion of the esophagus is not seen because it has a tendency to become invaginated into the fundus of the stomach and because it is covered by the extreme portion of the left lobe of the liver. As previously described, this portion of the liver is attached to the diaphragm by the

left triangular ligament. The first step in the mobilization of the esophagus is to incise this ligament as illustrated in Figure 402, *a*. This permits retraction of the liver laterally and away from the esophagus. Retraction of the liver is done either with a broad Deaver or malleable flat retractor. Since the ligament is avascular, its division may be accomplished with scissors without use of hemostats.

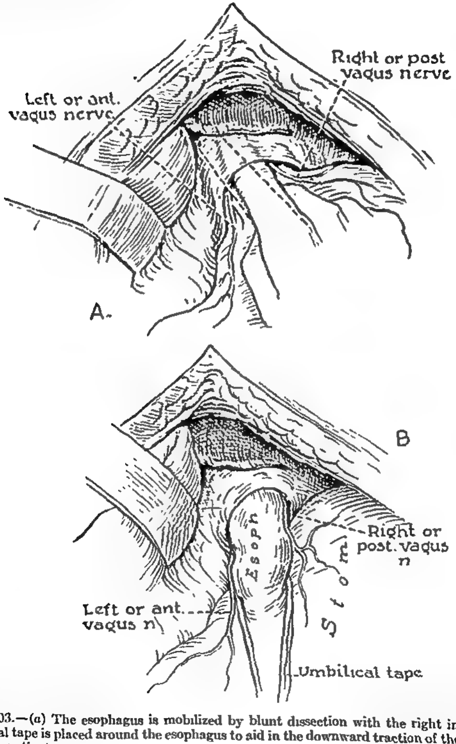

Fig. 403.—(*a*) The esophagus is mobilized by blunt dissection with the right index finger; (*b*) umbilical tape is placed around the esophagus to aid in the downward traction of the esophagus n the obese patient.

After dividing the triangular ligament, the stomach is grasped and drawn downward until the esophagus is placed on a stretch. The esophagus is now [illegible]

[illegible] before the operation. This also serves to keep the stomach

empty. The esophagus is now grasped with the left hand and the *peritoneum* is incised at its reflection with the diaphragm. Using the right index finger, the esophagus is mobilized by blunt dissection and pulled downward into the abdomen (Fig. 403, *a*). A strip of umbilical tape may be placed around the esophagus to aid in pulling it downward when the patient is obese or when mobilization is difficult. In this manner the esophagus may be gradually pulled downward for a distance of at least 10 centimeters (Fig. 403, *b*).

Excision of Vagi.—After the esophagus has been mobilized, the left vagus nerve is identified by its cord like consistency. Its relationship to the esophagus varies greatly, however, in the great majority of cases it is found anterior and somewhat to the left. As soon as it is identified, it is picked up with a long nerve hook and by

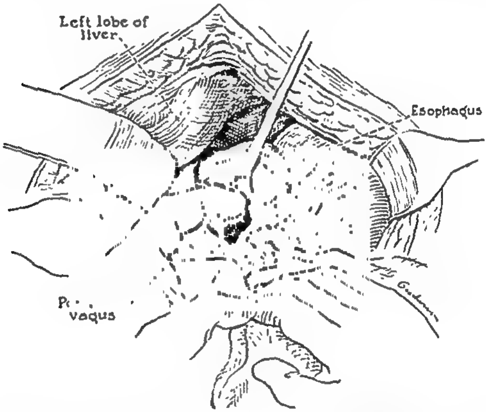

Fig. 404.—Posterior vagus nerve is hooked over the index finger and brought forward from its position posterior to the esophagus. (Redrawn after Crile, Jr.).

blunt dissection it is freed up to or through the hiatus of the diaphragm. It is important to isolate the nerve as high up as possible so that when the nerve is divided its proximal end will retract into the mediastinum. The nerve is now clamped, divided and tied with either silk or cotton and the proximal end is permitted to retract into the mediastinum. The lower end of the nerve is cut as far down as possible and the excised portion is discarded.

After the left vagus has been excised, the right nerve is located by palpation as a tense cord lying on the posterior surface of the esophagus. This nerve is larger than the left, and for this reason no difficulty should be encountered in its identification. The nerve is now isolated by finger dissection and it is then hooked over the index finger and withdrawn from its retroperitoneal position (Fig. 404). The nerve is then picked up with a long nerve hook and bluntly dissected up to or through the esophageal hiatus, and treated as the left vagus.

A final inspection is made to make certain that the esophagus is clear of all nerve fibers for a distance of 2 to 3 centimeters and to divide all the fibers passing to the stomach through the diaphragm. The peritoneal reflection over the esophagus may now be sutured, although this is unnecessary. Nor is it necessary to suture the triangular ligament to the diaphragm, unless this can be done with ease. After the vagal neurectomy has been done, the surgeon performs any additional gastric operation if indicated, and the abdomen is then closed without drainage in the usual fashion.

SUPRADIAPHRAGMATIC VAGAL NEURECTOMY

Endotrachial anesthesia is used. The anesthesia must be administered by an expert who thoroughly understands pulmonary physiology. Complete collapse of the lung must be avoided. The incidence of postoperative complications depends in a large measure on the skill of administration of the anesthesia.

The patient is placed on the right side with the head resting on the abducted arm and the patient's back close to the edge of the operating table. The left arm is held forward with the hand in front of the face. Sand-bags may be placed against the anterior portion of the chest to prevent the patient from rolling forward. The left leg is flexed on the thigh over the outstretched right leg.

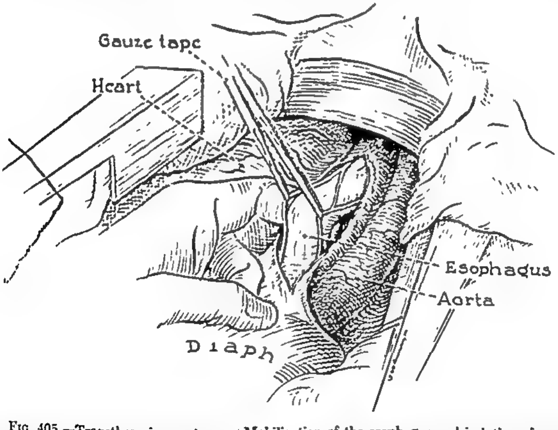

FIG. 405.—Transthoracic vagotomy. Mobilization of the esophagus and isolation of vagus nerves showing the communication between the anterior and posterior trunks. (Redrawn after Dragstedt.)

The following closely describes Dragstedt's technique of vagal neurectomy. An incision is made over the eighth left rib and the rib is extensively resected sub-periostially together with a portion of the cartilage. The left pleural cavity is then entered by incising the bed of the eighth rib. The intercostal nerve is

isolated at the posterior margin of the wound, divided and ligated with silk or cotton. This procedure minimizes postoperative pain in the chest. Adequate exposure of the thoracic cage is obtained by inserting a rib retractor.

The lung will be found collapsed when the pleura is opened, but complete collapse must not be permitted to take place. The inferior pulmonary ligament is divided and the left lung is retracted superiorly. A vertical incision, about 4 inches long, is made in the mediastinal pleura covering the lower end of the esophagus. The pleural edges are dissected away from the underlying esophagus. With blunt dissection the lower end of the esophagus is mobilized and lifted up into the pleural cavity (Fig. 405). Small esophageal arteries arising from the aorta should be ligated. The phrenic nerve is pushed to one side. The diaphragm is held down with a retractor.

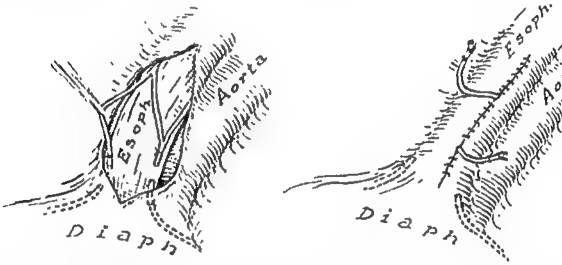

FIG. 406.—(a) Ligature and division of the vagus nerves just above the diaphragm; and (b) transplantation of proximal ends of cut vagi. (Redrawn after Dragstedt.)

The vagus nerve trunks are easily identified by palpation and dissection as indicated in Figure 405. The surgeon is referred to the surgical anatomy of the vagi for the various arrangements of the nerves on the esophagus. After carefully identifying the nerve trunks, they are picked with long nerve hooks and isolated for a distance of 1 or more inches. It is important that the nerves are freed as far as possible in the esophageal opening of the diaphragm. The nerves are now clamped, divided and tied with silk or cotton. The distal ends are permitted to retract into the abdomen and the proximal ends are sutured to the pleura. Dragstedt divides the nerves, without excising, and sutures the proximal ends to the pleura as illustrated in Figure 406. In this manner regeneration of the nerve is hindered. If possible, portions of the nerves should be excised.

The esophagus is now gently placed back in its bed and the pleura is closed with a running plain catgut stitch. The chest is usually closed without drainage

Postoperative Treatment.—The most serious complication of vagus neurectomy is gastric dilatation. According to Dragstedt, this is due to the accumulation of swallowed air and gastric and intestinal secretions. Occasionally, it may also be due to overdistention of food and drink. If overdistention of the stomach is prevented, a readjustment in the peripheral motor mechanism takes place, so that in a relatively short time the motor function of the stomach returns to normal.

A Levine tube is introduced into the stomach from twenty-four to forty-eight hours before the operation and left in place for four to five days after the operation. Suction is applied by the Wangensteen apparatus and the stomach is kept completely decompressed. When the tube is removed on the fourth or fifth day, the patient is permitted to take 30 cc. of water per hour for the first day. At the end of the day, the stomach is aspirated, and if empty, 60 cc. of water is permitted for the following day. If aspiration reveals that this larger amount has left the stomach, the patient is placed on a clear liquid diet for two or three days. The patient is instructed to cease eating if he feels distended. If the patient tolerates fluids easily, semi-solid food is gradually added. Dragstedt has found that some patients have returned to a full diet within two to three weeks without ill effects, others voluntarily restrict food intake for five to six weeks. He further states that if the stomach has not become overdistended in the immediate postoperative period, it is likely that most patients regain normal gastric function within six months.

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CHAPTER 56

GASTRIC SURGERY—GASTROSTOMY

By A. V. Partipilo

GENERAL CONSIDERATIONS

Gastrostomy consists of making an artificial opening into the stomach, through which a patient may be fed when food cannot be taken in the ordinary way. Conditions which prevent feeding through the usual route are: (1) cancer of the esophagus, causing obstruction to swallowing of food; (2) intractable stenosis of the esophagus; (3) cicatricial strictures, due to healing of ulcers produced by the swallowing of corrosive chemicals; (4) large esophageal diverticulum interfering with swallowing; (5) benign or malignant tumors of the cardiac portion of the stomach; and (6) as a route of approach for the treatment of cardiac stenosis, and for extra-esophageal tumors producing pressure on the esophagus.

Numerous methods have been described which for some reason or other have failed to give satisfactory results. According, to Quick and Martin ("Common Methods of Gastrostomy," Surg., Gynec. and Obst., March, 1928, p. 426), an ideal gastrostomy must have the following characteristics:

1. The fistula must have a lining not affected by the action of the gastric juices. The only such lining is gastric mucosa.

2. The fistula must be permanent, requiring neither the presence of a tube between feedings nor its presence over longer intervals, should the necessity for a gastrostomy be obviated by a temporary relief from dysphagia.

3. The fistula must be continent and permit no leakage of either gastric juices or of necessary liquid ingesta.

4. The fistula must permit easy and repeated instrumentation, such as gastroscopy, retrograde esophagoscopy, or retrograde bouginage.

5. The fistula must permit easy insertion of the tube so that the patient may feed himself.

6. The operation must offer as its end-result no unhealed or granulating surface subject to infection.

7. The operation must be possible in either a contracted or diseased cardia.

WITZEL'S METHOD

Make a left paramedian incision about 3 inches long, starting at the costal margin. The abdominal cavity is entered, and the anterior wall of the stomach is brought into the wound. Make a small submucus incision on the anterior surface of the stomach (Fig. 407). The mucosa is then grasped with an Allis forceps and cut. Through this opening [illegible] rubber catheter for a distance of 2 inches. Max Thorek, in his book "Surgic[illegible] Safeguards," cautions against placing the tube into the stomach more [illegible] 6 centimeters, so as to avoid kinking of the tub[illegible] gastric ope[illegible] snugly around the rubber tube.

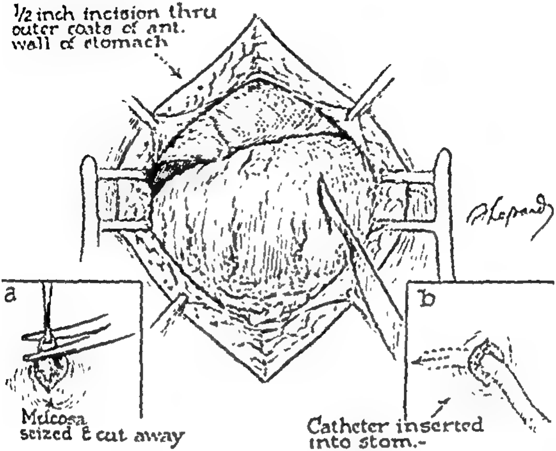
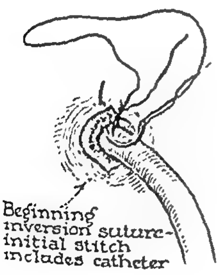


FIG. 407.—Witzel's gastrostomy. Incision through outer coats of the stomach: (a) an opening is made into the bulging mucosa; (b) tube is inserted through the opening.



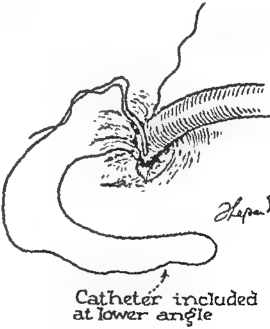


Fig. 408

Fig. 409

FIG. 408.—A Lembert stitch attaches the tube to the gastric opening.

FIG. 409.—Lembert stitches applied to invert the gastric wall around the tube. When the halfway point is reached another Lembert stitch is taken through the tube.

The first stitch anchors the tube, then a series of continuous Lembert stitches are applied on the gastric wall only, until the opposite side is reached, when another anchor stitch is taken through the tube (Figs. 408 and 409).

After the gastric opening has been sutured, the tube is laid on the stomach for a distance of 1½ inches, and a gutter is made for it by raising a fold of stomach on either side and suturing the fold over the tube (Fig. 410). Interrupted or continuous sutures may be employed for suturing the folds. The author prefers the continuous Cushing suture because it is easier to apply and it affects a more secure and accurate approximation of the folds.

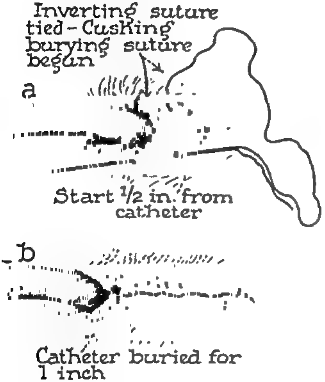


Fig 410

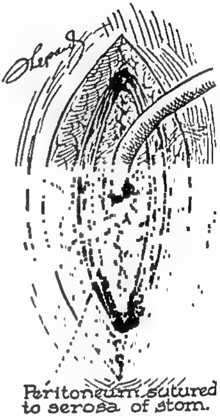


Fig. 411

FIG. 410—The tube is buried within the gastric wall with a continuous suture.
FIG. 411.—The peritoneum is sutured to the stomach wall around the rubber tube.

As a final step the parietal peritoneum is sutured to the stomach wall immediately around the rubber tube (Fig. 411). An advantage of the Witzel Method is its technical simplicity, requiring only a few minutes to perform the operation. A most efficient valve is formed by the oblique tract. When the stomach is distended the walls of the oblique tract are forced together, thereby preventing leakage.

Since the "gutter" is lined with serosa, the walls will quickly adhere if the tube should be pulled out accidentally. For this reason reinsert the tube as soon as possible.

INTRAMURAL GASTROSTOMY

Soresi devised a simple gastrostomy by providing a passageway for the catheter through the wall of the stomach. The passageway had two openings which were situated perpendicularly to the two extremes of the stomach. In collaboration with

C. W. Frey, of York, Pennsylvania, the operation was modified by the author to overcome some of the technical difficulties and objectionable features which were encountered with the original Soresi method.

Under local or light gas anesthesia, a left paramedian incision is made from the costal margin downward for a distance of 2 or 3 inches. The abdominal cavity is entered and the stomach is delivered into the wound. Beginning close to the greater curvature, an incision is made through the serosa and muscularis exposing

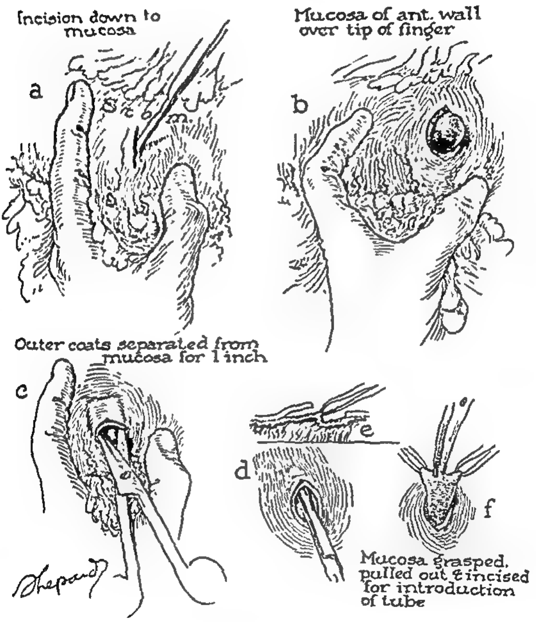


FIG. 412.—Intramural gastrostomy.

the mucosa for a distance of about $\frac{3}{4}$ of an inch (Fig. 412, *a*, *b*). The direction of the incision is toward the lesser curvature. Using a small Mayo dissecting scissors, separate the muscularis for a distance of 1 inch (Fig. 412, *c*). At the extreme end of the separation the underlying mucosa is picked up with Allis forceps (Fig. 412, *d*, *e*), a small opening is made, and a catheter is introduced through it. The tube is anchored to the edges of the opened mucosa, and then pushed back under the muscularis as far as it will go. That portion of the tube lying over the bulging

mucosa is now buried by suturing the margins of the incised stomach over it (Fig. 413, *b*, *c*, *d*). The first stitch anchors the tube to the external outlet of the tunnel (Fig. 413, *b*).

The tube may be brought outside through a nearby stab wound, or can emerge through the original incision. In any event the peritoneum is sutured to the stomach wall around the point of emergence of the tube. Feeding may be started as soon as the patient returns to bed.

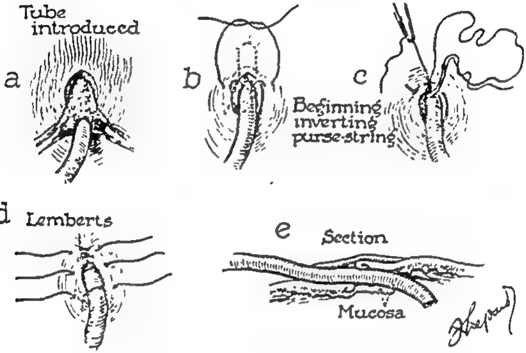


Fig. 413.—Intramural gastrostomy. Application of tube; (*e*) illustrates the scheme in the formation of valve and the intramural position of the tube.

FRANK'S GASTROSTOMY

Make an oblique incision parallel to the left costal margin. The length need not exceed 3 inches. A cone-shaped portion of the stomach is withdrawn and held forward until the base of the cone is sutured to the peritoneum. A second incision is made through the skin, about 1 to 2 inches below and parallel to the longer upper incision. Separate the subcutaneous tissue between the two incisions so as to connect the two openings beneath the subcutaneous tissue and skin. An Allis forceps is passed beneath the smaller incision and grasps the apex of the stomach cone, which is then drawn underneath the bridge and beyond the surface of the second incision, where it is attached with a few interrupted sutures. After this has been accomplished, make a small opening into the apex of the protruding cone and suture its edges to the skin. A catheter is introduced through the opening, and the patient can be fed through this tube as soon as desired.

In this method a valvular opening is made which functions satisfactorily when the operation is properly done. The objection to the method is that in carcinoma of the esophagus the stomach may become so contracted that not enough gastric wall is available for the formation of the cone.

KADER'S METHOD

The abdomen is opened through a paramedian incision and the stomach is delivered through the wound. A small opening is made into the anterior surface of the stomach, and a tube is introduced and fixed to the margins of the opening.

As illustrated in Figure 414, two parallel folds of stomach are raised, one on each side of the tube, and are sutured with interrupted Lembert stitches. The same number of sutures are applied above and below the tube. Two additional parallel folds are raised and sutured. The operation is completed by suturing the stomach to the anterior parietal peritoneum. In this method an efficient valve is formed by the two tiers of folded stomach wall on either side of the tube, thus preventing leakage. If necessary, another tier may be raised to increase the valvular action.

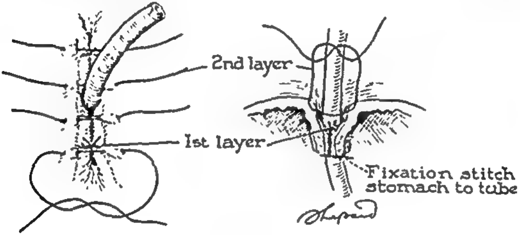


Fig. 414.—Kader's gastrostomy.

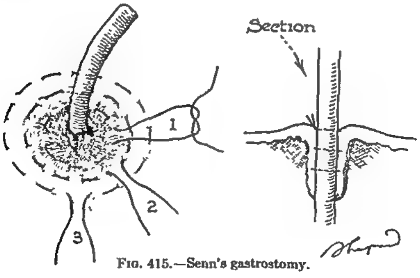


Fig. 415.—Senn's gastrostomy.

SENN'S METHOD

Senn's modification consists of infolding the tube in the stomach with two or three purse-string sutures. The first purse-string suture includes an anchor stitch in the tube. The sutures are applied about ½ inch apart and the tube is pushed inward while each suture is being tied. The result is that the tube will lie within a funnel-shaped inverted stomach wall. Senn's modification has the advantage because it is

easy to perform, less stomach wall is utilized for the formation of the valve, and a valve is formed which satisfactorily prevents leakage. As in the Witzel and Kader methods, the stomach immediately around the point of emergence of the tube is sutured to the anterior parietal peritoneum.

BRUNSCHWIG'S METHOD

Brunschwig's method is a modification of Senn's gastrostomy. In the palliative treatment of carcinoma of the esophagus and carcinoma of the upper portion of the stomach with extension into the esophagus, Brunschwig performs a "de Pezzer Catheter Gastrostomy" followed by intensive x-ray treatment of the lesion. During the period of irradiation and following it the patients are nourished entirely through the gastrostomy. If, following the subsidence of the skin reaction from irradiation, the patient can gradually return to nourishing himself entirely by mouth, the gastrostomy tube is withdrawn.

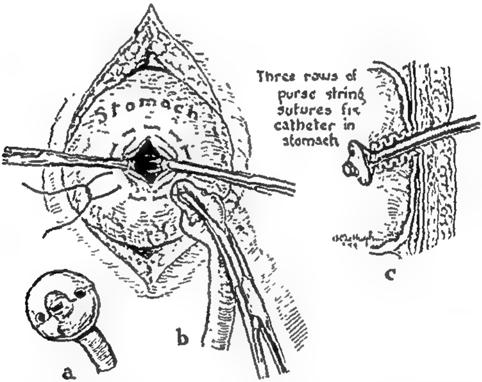


FIG. 416.—Brunschwig's gastrotomy: (a) large phalange de Pezzer catheter, size 20 F; (b) insertion of catheter into stomach; (c) diagrammatic representation of catheter in place after completion of operation. (Courtesy of Dr. Brunschwig and Amer. J. Surg.)

The technical steps of the method are illustrated in Figure 416. A high midline incision is made under local anesthesia. The length of the incision depends upon whether the abdomen is to be explored; if not the incision need not be longer than 4 inches. The stomach is brought out through the incision and a purse-string suture is inserted about the site of the opening in the stomach. This is usually done at the midway point between the curvatures. The stomach wall is then perforated in the center of the purse string. The phalange of a large de Pezzer catheter is now compressed by a large Kelly forceps, and inserted into the stomach (Fig. 416, b). The forceps is withdrawn and the purse-string suture is tied firmly about the catheter. The stomach wall is then further invaginated about the catheter by two or more successive purse-string sutures (Fig. 416, c).

The stomach wall about the catheter is now sutured to the parietal peritoneum with interrupted silk or cotton sutures, and the abdominal incision is then closed in the usual manner. Sterile strips of gauze are tied about the catheter and pressed firmly down upon the skin.

JANEWAY'S GASTROSTOMY

The principle of Janeway's gastrostomy is the formation of a "goose-neck" from a flap of the anterior wall of the stomach. When a permanent gastrostomy is indicated this method is undoubtedly the operation of choice.

The abdomen is opened through a mid-line incision extending between the ensiform cartilage and the umbilicus. After the abdominal cavity has been entered, the stomach is delivered into the abdominal wound. Figure 417 illustrates various

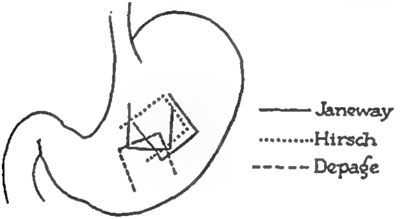


FIG. 417.—Various incisions used for Janeway's gastrostomy.

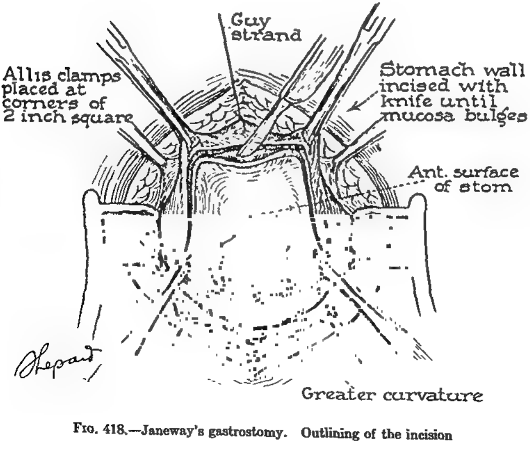


FIG. 418.—Janeway's gastrostomy. Outlining of the incision

incisions which may be selected for the proposed flap. An incision made with the base of the flap attached close to the greater curvature will least disturb the blood supply to the flap. The proposed site of the flap on the anterior wall of the stomach should be chosen as high up on the cardia as possible.

After the proposed site for the flap has been determined, the abdominal cavity is walled off and protected from any possible spilling of gastric content. As illustrated in Figure 418, a flap is outlined with its base at the greater curvature and its free end at the lesser curvature. Two Allis forceps are placed about 2.5 centimeters apart and as close as possible to the lesser curvature. Two additional forceps are applied about 6 centimeters directly opposite, close to the greater curvature, to mark the base of the flap. At this time superficial vessels leading into the proposed line of incision are secured.

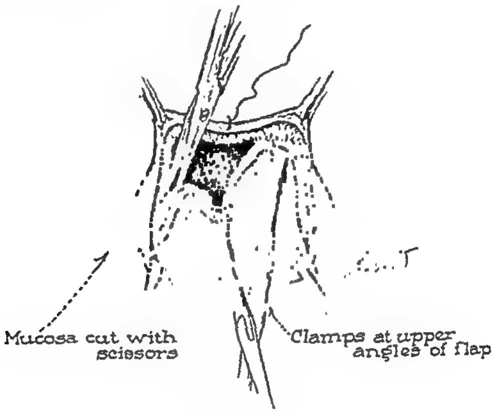


Fig. 419.—Completing the incision with scissors

Using a scalpel, an incision is made on the serosa between the two Allis forceps at the lesser curvature, and between these and the Allis forceps close to the greater curvature (Fig. 418). The incision is then deepened until the mucosa bulges, thus exposing large vessels which are stick-tied with plain catgut No. 00. After this has been accomplished, a guy suture is inserted in the middle of the incision along the lesser curvature, a stab wound is made, and the mucosa is completely incised with scissors (Fig. 419). Notice that two additional Allis forceps are applied at the corners of the free flap. When the mucosa is first opened, gastric juices are removed with a suction apparatus, and every precaution employed to prevent contaminating the abdominal cavity.

After all bleeding points have been ligated, the flap is turned down, the pyloric sphincter is relaxed with gentle manipulation, and a soft rubber tube 25 centimeters long and 4 millimeters in diameter is passed through the sphincter well into the

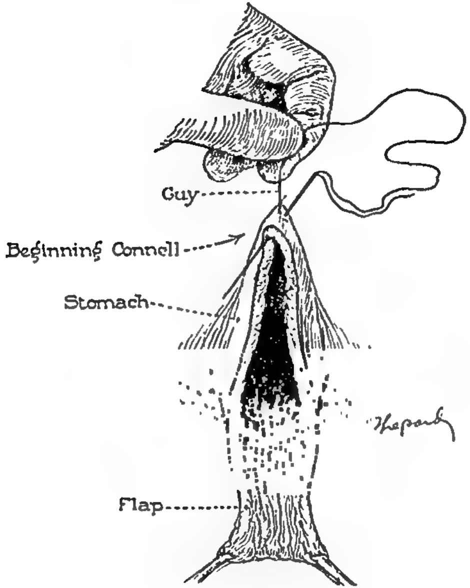


Fig 420.—Beginning of Connell suture for the closure of the stomach edges.

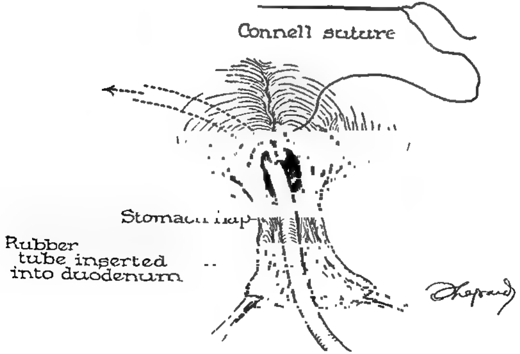


Fig. 421.—Rubber tube inserted into the stomach before the "gooseneck" is closed

duodenum. The free end of the tube is temporarily fastened with a clamp to the end of the flap. Quick and Martin use a 12 F catheter, and never use a catheter larger than a 16 F. They believe that a small size catheter aids in the control of leakage.

The wound in the stomach is closed with a Connell stitch. This is begun in the middle of the lesser curvature (Fig. 420), and when the base of the flap is reached it is continued on to the extremity of the flap (Fig. 421), thus producing a "goose-

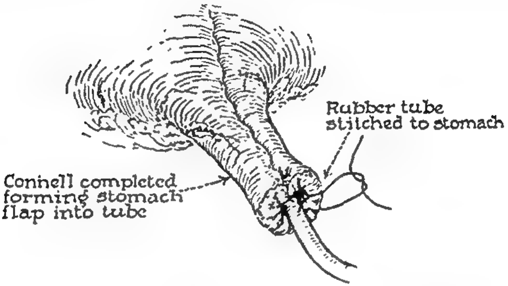


Fig 422.—Connell suture completed thus forming a gooseneck from the stomach flap

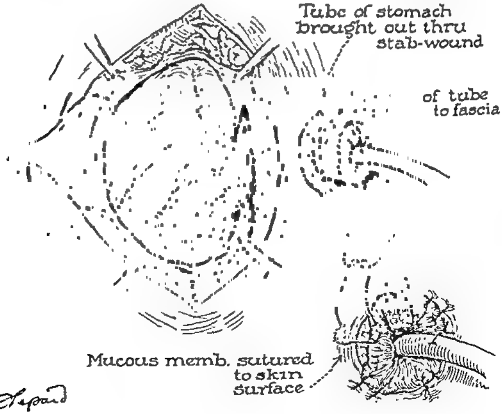


Fig. 423

Fig. 424

Fig 423.—The end of the gooseneck is brought through a stab wound. Interrupted stitches anchor the serosa of the gooseneck to the fascia.

Fig. 424.—The mucosa is stitched to the skin.

neck" from the stomach flap. The suture is tied and the ends are cut. An interrupted stitch secures the catheter to the mucosa at the margin of the goosneck, and the ends are left long (Fig. 422). A second row of Cushing stitches is applied to reinforce the suture line. Make a stab wound about 2.5 centimeters long through the outer border of the costal margin. The incision is depened through the rectus muscle and fascia. Introduce a large Kelly forceps through the stab wound and grasp the catheter and the long ends of the sutures which secure the tube to the goose-neck, and, assisted by gentle manipulation from within, the goose-neck

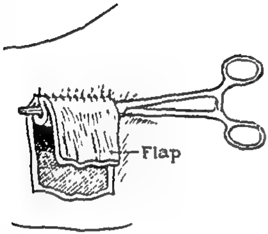


Fig. 425

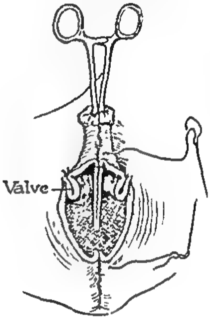


Fig. 426

FIG. 425.—Spivack's modification of Janeway's gastrostomy.

FIG 426.—Schematic drawing illustrating the formation of a valve at the base of the goose-neck.

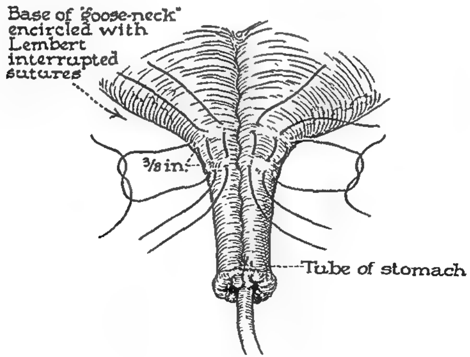


FIG. 427.—Author's method of forming Spivack's valve. The base of the gooseneck is encircled with a series of Lembert stitches.

with the catheter is pulled through the stab wound. As illustrated in Figure 423 the serosa of the goose-neck is fastened to the anterior rectus sheath with a few interrupted chromic catgut No. 1 stitches. The mucosa is sutured to the skin with silk stitches; avoid including the serosa or muscularis (Fig. 424). The tube is secured to the mucosa and after a final inspection of the suture line in the stomach, the original midline incision is closed in the usual manner. Dressings are applied so that the rubber tubing is carried through all the bandages. The rubber tube is clamped until ready for use, and adhesive plaster is placed around it to prevent undue tension.

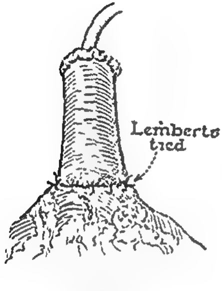


Fig. 428

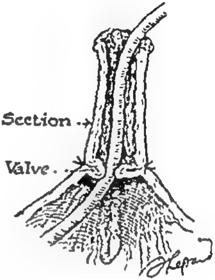


Fig. 429

FIG. 428.—Lembert stitches tied.
FIG. 429.—Schematic drawing illustrating the formation of a valve at the base of the gooseneck.

Feeding is begun as soon as the patient returns to bed. The following diet is suggested by Quick and Martin: Three ounces of peptonized milk are introduced every three hours on the day of the operation, and the amount is increased to 4 ounces every three hours the day following the operation; 3 ounces on the next day and so on until there is definite discomfort after feeding, indicating that the capacity of the stomach has been reached. For a few days saline is given per hypodermoclysis, intravenously, and by proctoclysis. The tube is shortened daily about 1 inch until the sixth day when it is entirely removed. It is reinserted whenever the patient is fed. Solids, such as eggs, butter, sugar, and other semi-solids may be introduced through the tube after the sixth day.

SPIVACK'S METHOD

According to Quick and Martin leakage occurs in only about 5 per cent of cases. In order to avoid leakage, Spivack described a modification of the Janeway by making a tubular valve at the base of goose-neck. The valve, while not interfering with the introduction of food, prevents the escape of gastric juices by closing hermetically the stomach opening at the base of the gooseneck.

Figures 425 and 426 illustrate the characteristic steps for the formation of the tubular valve according to Spivack's technique. Figure 427 illustrates the author's method of making the valve after the flap has been entirely inverted. A series of

interrupted Lembert sutures are applied around the base of the gooseneck, and when tied (Fig. 428) a tubular valve is formed as shown in Figure 429.

BECK-JIANU METHOD

The Beck-Jianu gastrostomy, as described by Sweet of Boston, consists of a tube constructed from the stomach wall along the greater curvature. Sweet believes that this method is more comfortable and easier to care for than is the older catheter method.

A left upper quadrant muscle splitting incision is made through the rectus muscle. The abdominal organs are explored and the stomach is brought out into the wound. The right gastro-epiploic vessels are ligated and cut near the antral

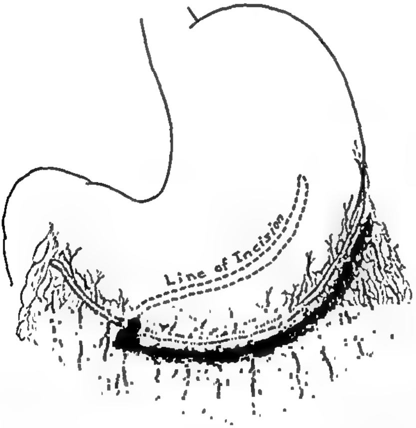


Fig. 430.—Beck-Jianu gastrostomy. Division of the gastrocolic omentum; ligation of the right gastro-epiploic vessels. Dotted line shows direction and optimal length of incision through the stomach walls. (Courtesy of Dr. Sweet and Surg. Gynec. and Obst.)

portion of the stomach (Fig. 430). The gastrocolic ligament is then divided from this point as far to the left as is necessary to permit the construction of a tube of about 5 to 6 inches long. The left gastro-epiploic vessels are retained to carry the blood supply to the newly constructed tube. Two long, curved, flexible gastric clamps are now placed on the stomach parallel with the direction of the greater curvature of the stomach. Sweet emphasized that the proposed incision on the stomach must be kept parallel with the greater curvature, otherwise, an hour-glass deformity with malfunction because of obstruction will result. The stomach is now incised between the two gastric clamps for the desired length. The handles of the

clamps are separated and the flap is swung away from the body of the stomach (Fig. 431).

The edges of the stomach are now sutured with either a Connell stich or in layers. The closure of the stomach is begun on the body of the stomach at the antral end and is continued upward to the end of the gastric tube.

If a Connell suture was used, a second outer row of Cushing stitch is now applied. After this, the cut edge of the gastrocolic ligament is sutured to the newly-formed greater curvature (Fig. 432).

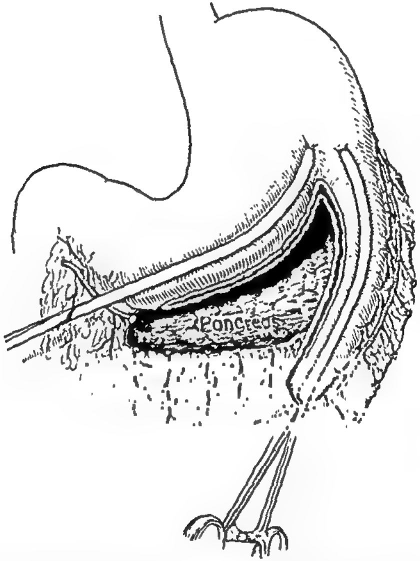


FIG. 431 —Beck-Jianu gastrostomy. Stomach divided and ready for suture. Note the preservation of the left gastro-epiploic vessels on the gastric tube Clamps shown in place as actually used during the operation. (Courtesy of Dr. Sweet and Surg, Gynec. and Obst.)

Now make a short transverse skin incision on the anterior chest wall several inches above the upper end of the abdominal incision. With finger dissection, a channel is made anterior to the rectus fascia between this new incision and the abdominal wound. The gastric tube is then brought out of the peritoneal cavity, passed out through the subcutaneous channel and out through the transverse incision (Fig. 433). The gastric tube must be drawn out tightly around the costal margin and pulled as high as possible on the chest wall.

GLASSMAN'S METHOD

The abdomen is opened through a high left upper mid-rectus incision and the stomach is exposed. The anterior wall of the stomach is grasped with a Babcock forceps at the most mobile point, which is usually located midway between the fundus and the antrum and equi-distant from the greater and lesser curvatures. With upward traction, the stomach wall is converted into a cone-shaped diverticulum.

Using braided silk, a purse-string suture is applied at the base of the cone (Fig. 434, *a*). The purse-string is tensed and tied so that the base is puckered and the lumen at the base of the cone is almost but not completely constricted. Glassman has found that it is not essential for the purse-string suture to be perfectly circular about the base of the cone. An oval or irregular shaped purse-string suture forms a diverticulum entirely satisfactory. The Babcock forceps may now be released and re-applied. A second re-enforcing purse-string is now applied about ¼ of an inch above the first (Fig. 434, *b*). A third purse-string is introduced around the diverticulum about ¼ of an inch above the second (Fig. 434, *c*).

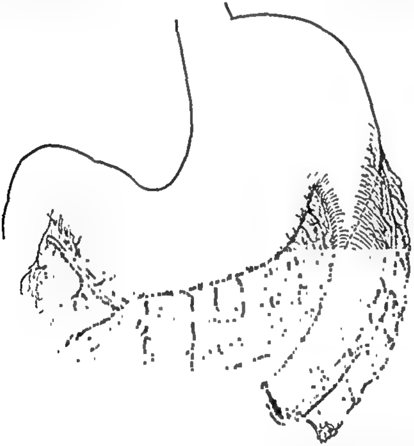


FIG. 432.—Beck-Jianu gastrostomy. [illegible] body of the stomach and gastric tube compl[illegible] mach. Gastric tube lies free and ready to be [illegible] Dr. Sweet and Surg., Gynec. and Obst.)

A series of interrupted Lembert stitches, usually eight to ten, are now inserted into the seromusculature of the stomach below the three purse-string sutures, and passed above (Fig. 434, *c*). When these are tensed and tied, a circular valve is created at the base of the tube.

Fig. 433.—Beck-Jianu gastrostomy. Relative position of the gastric tube after completion of the operation. The tube has been brought out around the costal margin, up through a subcutaneous tunnel on the anterior chest wall and out through a small transverse incision several inches above. *(Courtesy of Dr. Sweet and Surg., Gynec. and Obst.)*

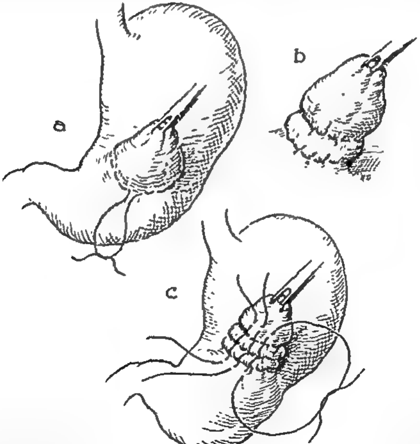


Fig. 434.—Glassman's double-valve tubogastrostomy: (*a*) formation of a cone-shaped diverticulum from the anterior wall of the stomach by traction; (*b*) fixation of the formed diverticulum by two purse-string sutures; (*c*) insertion of the third purse-string suture approximately midway between the first purse-string and the apex of the diverticulum.

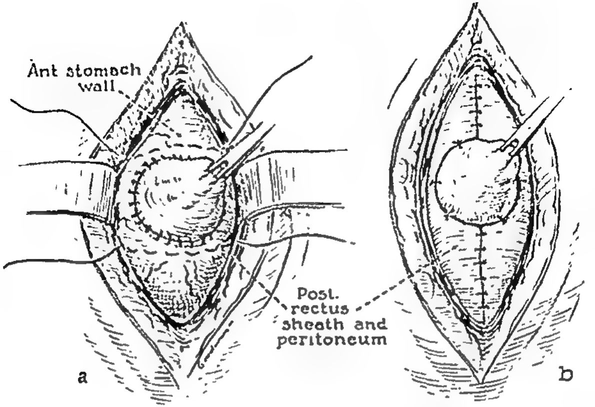


Fig. 435.—Glassman's method: (*a*) anchoring of the anterior stomach wall to the peritoneum and posterior rectus sheath; (*b*) the posterior rectus sheath is sutured snugly around the base of the tube.

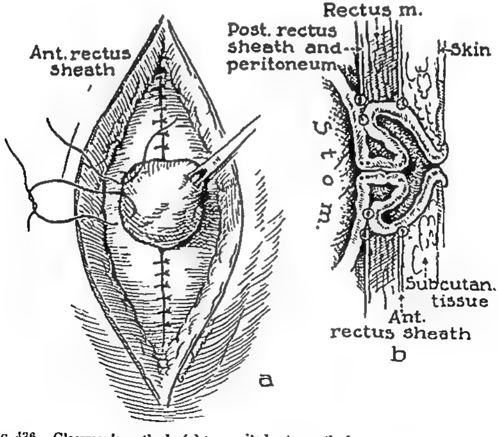


Fig 436 —Glassman's method· (*a*) two united sutures, the lower one representing the suture that attac
to furthe
the first .
valves

Interrupted sutures are now inserted into the anterior wall of the stomach at points superior, inferior, and lateral to the base of the gastric cone in order to fix the stomach wall to the peritoneum and posterior rectus sheath (Fig. 435, *a*). In a similar manner the tube is attached to the anterior rectus sheath (Fig. 436, *a*). The skin edges around the tube is closed in the usual fashion. Whenever the tube does not reach above the level of the skin, the skin edges may be inverted and attached to the anterior rectus sheath just where the apex of the tube emerges to the surface. Figure 436, *b* illustrates the arrangement of Glassman's aseptic tubovalvular gastrostomy.

The opening into the apex of the tube, by means of a cautery, may be done at any time from the first to the tenth postoperative day (preferably the fifth). By delaying the opening as long as possible, an aseptic tubovalvular gastrostomy is achieved which distinguishes this method. Regular tubal feedings begin after the stoma is created.

Questionnaire on page 625.

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CHAPTER 57

GASTRIC SURGERY—PYLOROPLASTY

By A. V. Partipilo

GENERAL CONSIDERATIONS

The first pyloroplastic operation for the relief of pyloric obstruction was probably done by Loreta in 1882. He produced digital dilatation of the constricted pylorus. In 1886, Heineke, and in 1887, Mikulicz independently devised a pyloroplasty which consisted of dividing the pyloric stricture by making an incision parallel with the axis of the lumen and closing the opening in a transverse direction. This operation is now known as the Heineke-Mikulicz pyloroplasty. In 1907, Fredet devised a plastic operation somewhat similar to the Heineke-Mikulicz. He made a longitudinal incision through the various coats down to the submucosa and then sutured the wound transversely. Five years later Rammstedt remarked upon the beneficial results of the Fredet operation, but he did not find it necessary to suture the wound. Many modifications have been suggested within recent times; however, it is generally conceded that for congenital hypertrophic pyloric stenosis Rammstedt's pyloroplasty gives satisfactory results.

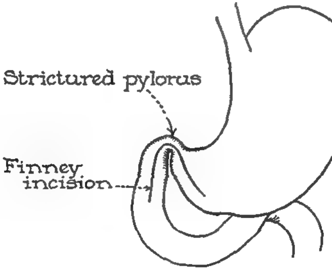


Fig. 437.—Horseshoe incision for Finney's pyloroplasty.

Another plastic operation is Finney's pyloroplasty. This consists of making a horseshoe incision on the pylorus and duodenum, and the wound *thus* made is closed in a manner to greatly increase the pyloro-duodenal orifice (Fig. 437). Finney's pyloroplasty is indicated in pyloric obstruction due to peptic ulcers. Of the many plastic operations, Finney's pyloroplasty has the widest application, and in properly selected cases it has distinct advantages over gastrojejunostomy. The feasibility of the operation depends upon whether or not the first portion of the duodenum can be mobilized. Because of technical difficulties Finney's operation has not

gained favor. At the present time posterior gastrojejunostomy is the operation of choice for pyloric obstruction due to peptic ulcers.

For ulcers on the posterior wall of the duodenum, Balfour's method of excising the ulcer combined with pyloroplasty has distinct advantages in selected cases. Partial gastrectomy has replaced local excision and pyloroplasty in this type of ulcer, nevertheless in certain cases when resection is not feasible nor desirable, Balfour's technique commends itself. The author has used this procedure for excising an "impending perforating" anterior duodenal ulcer when he did not feel justified in doing a partial gastrectomy.

RAMMSTEDT PYLOROPLASTY

This operation is most often indicated for congenital pyloric stenosis. The importance of preparing an infant for the operation cannot be overestimated. If the condition of the patient warrants, the operation may be postponed for a day or two during which time dehydration is corrected. For anesthesia the author utilizes a sugar pacifier saturated with whiskey, combined with local infiltration of

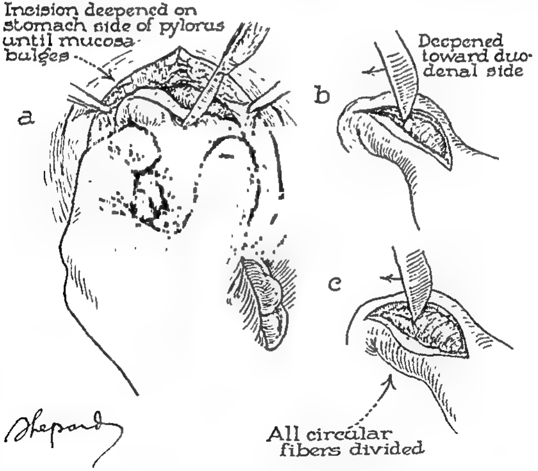


FIG. 438.—Rammstedt's pyloroplasty.

the abdominal tissues with ½ per cent novocaine. The objection to local anesthesia is that it causes straining of the infant, producing eventration. We have not found this objection sufficiently grave to discard local anesthesia. The use of whiskey produces adequate relaxation and warm packs may also be held firmly over the wound until the infant stops straining.

The abdomen is opened through a high right rectus incision made about 1½ inches long. As soon as the peritoneal cavity is entered, the pyloric tumor is examined and the mass is delivered *into the abdominal wound with care.* Infants do not stand shock and infection well. For these reasons the surgeon should use extreme gentleness in the handling of tissues, prevent loss of body heat from undue exposure, and affect a most careful hemostasis.

As illustrated in Figure 438, the pyloric mass is steadied with the left hand. An incision is now outlined on the serosa extending from the duodenum to the normal stomach beyond the hypertrophic pylorus. Beginning on the stomach end, the circular muscle fibers are cut with the dull end of the scalpel blade until the mucosa bulges. While cutting a "grating" effect is experienced which indicates that the fibers have not all been cut, hence the cutting should be continued until

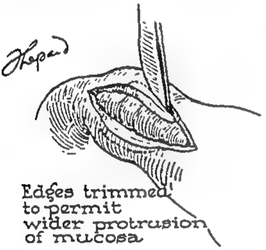


Fig. 439

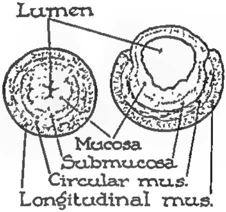


Fig. 440

Fig. 439.—Removing wedge-shaped piece of hypertrophied pyloric sphincter.

Fig. 440.—Scheme of Rammstedt's pyloroplasty.

this "grating" ceases. If the mucosa is puckered it means that some of the fibers have not been incised. When the duodenal margin is reached extreme caution must be employed to avoid opening the mucosa. However, it is rather important that the fibers are cleanly incised, otherwise relief will not be obtained. If the mucosa should be accidentally torn through, it is closed with fine silk. To permit a wider protrusion of the mucosa a wedge-shaped portion of the hypertrophied tissue may be trimmed away as illustrated in Figures 439 and 440.

As a rule no large vessel is encountered, and for this reason only a slight amount of oozing occurs which can be controlled by application of firm pressure with hot moist packs. Any large bleeder may be stick-tied with plain catgut No. 000. After all oozing and bledding has been controlled, the stomach is replaced into the abdomen without further reinforcement of the pyloroplasty.

If the mucosa has been torn, and to prevent adhesions, an omental graft can be placed over the bulging mucosa. Figure 441 illustrates the author's method of applying the omental graft. The omentum is thrown over the bulging mucosa and held in place with interrupted sutures, and the graft is then detached. It has been found that this procedure permits a better spreading of the graft than the method which sutures the graft after it has been detached.

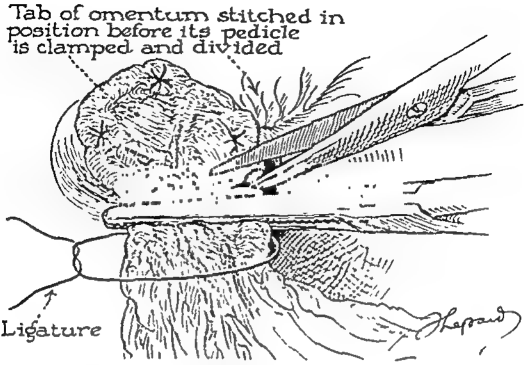


FIG. 441.—Author's method of applying an omental graft.

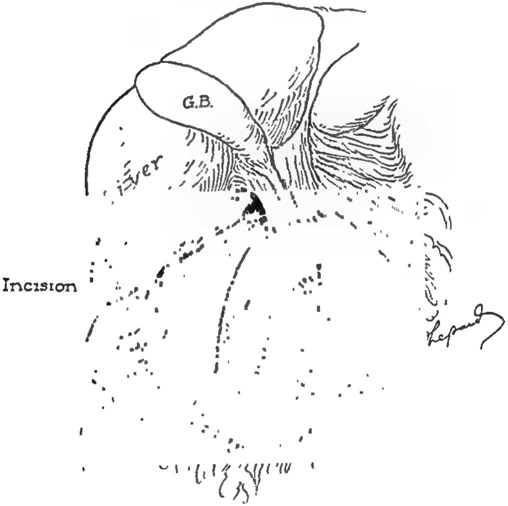


FIG. 442.—Kocher's incision to mobilize the duodenum.

After the operation the infant must be kept warm, but not so warm that he will lose his fluid by perspiration. Fluid replacement may be given as 5 per cent glucose hypodermically and by mouth. As soon as possible, the patient is placed on routine feedings. Care should be exercised to prevent soiling of the wound.

FINNEY'S PYLOROPLASTY

Technique.—The abdomen is opened through a right paramedian or Sloan's transverse incision. As previously stated the success of this operation and the ease and rapidity with which it can be done depends upon the mobilization of the duodenum. If examination reveals numerous adhesions binding down the duodenum and pylorus or a posterior ulcer that has perforated into the pancreas, it is better to decide upon some other procedure. On the other hand, if the duodenum can be mobilized easily, the operation is rather simple and can be performed without difficulty.

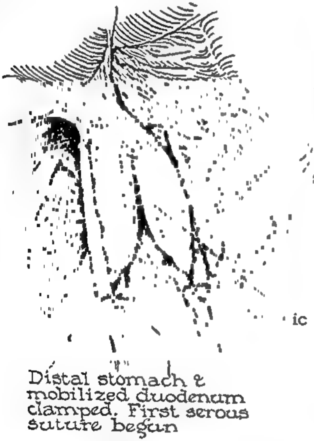


Fig. 443

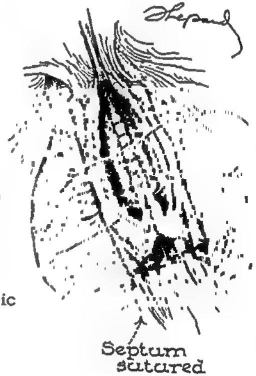


Fig. 444

FIG. 443.—Finney's pyloroplasty. The first outer row of suture is applied.

FIG. 444.—Finney's pyloroplasty. The septum is sutured with a continuous lock-stitch.

The duodenum is mobilized by Kocher's method. This is done by incising the parietal peritoneum vertically to the right of the duodenum (Fig. 442). With finger dissection, the duodenum is carefully lifted from its bed on the posterior body wall and drawn forward until it comes in contact with the stomach. If any difficulty is encountered, it may be due to a short hepatico-duodenal ligament which must be

incised carefully in order to complete the mobilization. The ligament should be excised only when it is certain that the underlying structures are separated from the ligament.

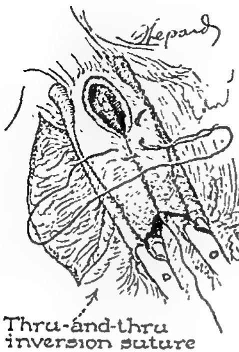


Fig. 445 Fig. 446

Fig. 445.—The corner has been inverted and the anterior edges are inverted with a Connell stitch.

Fig. 446 —The anterior outer row of Cushing suture.

After the duodenum and pylorus have been mobilized, intestinal clamps are applied to control the contents. Clamps may be dispensed with providing suction is used to collect the duodenal and gastric juices. The outer row of suture is started at the angle of the pylorus and continued as a Cushing suture approximating the duodenum and stomach for a distance of about 3 inches. Now make a horseshoe incision (Fig. 443), parallel with the posterior row of suture on the stomach, pylorus, and duodenum. The incision is made about ¼ inch from the suture line. All bleeding points are controlled. If the wall of the pylorus is thickened, or if it is contracted with scar tissue, it is excised. If the mucous membrane is extremely redundant, it is also excised. As illustrated in Figure 444, the septum is sutured with a continuous lock-stitch. When the angle of the stomach and duodenum is reached it is infolded, and the anterior margins are inverted with a Connell suture (Fig. 445). The operation is completed by applying an anterior outer row of Cushing sutures (Fig. 446).

Fig. 447.—Finney's pyloroplasty. Illustrates the enlarged lumen at the pyloric region

Finney originally employed interrupted mattress sutures for the anterior row, left them untied temporarily, and retracted them. The incision was then made, the posterior septum was sutured, and the interrupted sutures of the anterior outer row were tied. Continuous sutures have been used for the anastomosis because they are easily and quickly applied.

BALFOUR'S EXCISION-PYLOROPLASTY

Technique.—The abdomen is opened through a right paramedian or Sloan's incision. As in the case of Finney's pyloroplasty, the ease and rapidity with which this procedure can be done depends upon the degree of fixation of the duodenum. The duodenum is mobilized by Kocher's method, incising the parietal peritoneum vertically to the right of the duodenum as illustrated in Figure 442. With finger dissection, the duodenum is carefully lifted from its bed and drawn forward until

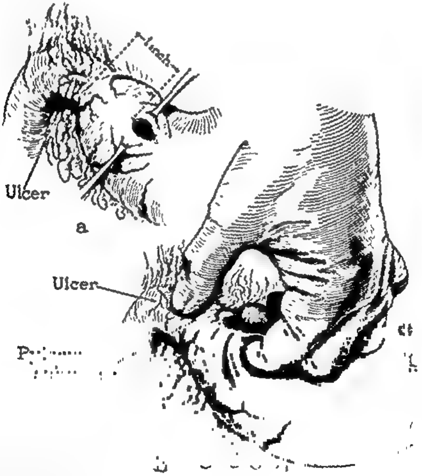


FIG. 448.—Balfour pyloroplasty. Exploration of duodenum. (Courtesy of Balfour and Surg., Gynec. and Obst.)

it comes in contact with the stomach. If any difficulty is encountered, it may be due to a short hepatico-duodenal ligament which must be incised carefully in order to complete the mobilization. The ligament should be excised only when it is certain that the underlying structures are separated from the ligament.

After the duodenum has been mobilized, the site of the inflammatory process in the anterior duodenal wall is marked off with Allis forceps. These are placed on the inferior and superior borders of the duodenum and slightly below the inflam-

matory process. Similar forceps are placed on the greater and lesser curvatures of the stomach, about 1 inch proximal to the pyloric sphincter. Two transfixing ligatures, one at the lesser and the other at the greater curvature side of the pyloric sphincter are applied (Fig. 449).

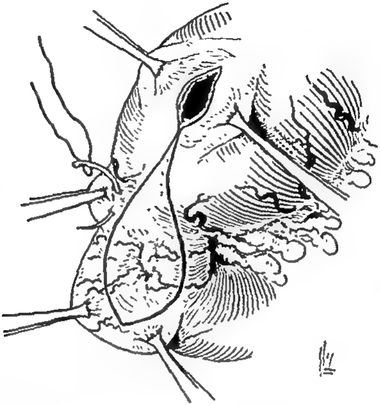


FIG. 449.—Excision of ulcer on anterior wall of duodenum. (Courtesy of Balfour and Surg., Gynec. and Obst.)

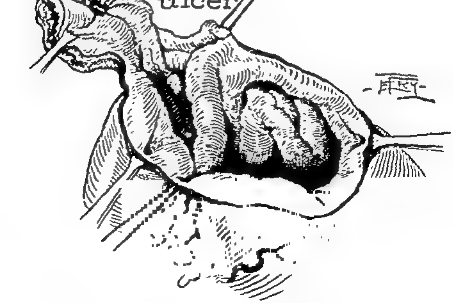


FIG. 450.—Excision of ulcer on anterior wall of duodenum. (Courtesy of Balfour and Surg., Gynec. and Obst.)

Now make an incision into the stomach about 1 inch above the sphincter and midway between the curvatures (Fig. 448, *a*). Introduce the index finger through this opening, through the pylorus, and carefully explore the posterior surface of the duodenum as illustrated in Figure 448, *b*.

If it becomes advisable to continue with the excision, the gastric incision is prolonged down through the pyloric sphincter to the inner side of the transfixing sutures (Figs. 449 and 450). The pyloric flap thus made is now examined and whatever lesion is present is excised by continuing the incision in the univolved portion of the anterior wall of the duodenum. The two tranfixing sutures can now be used as tractors.

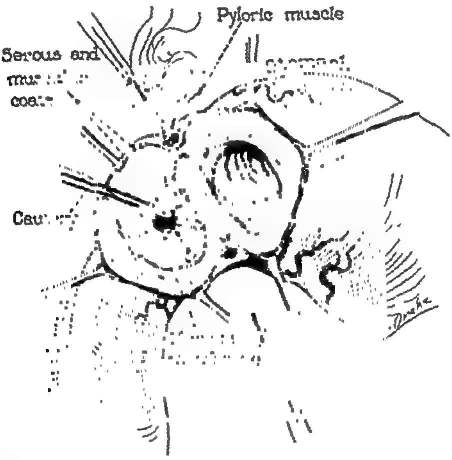


Fig. 451.—Cautery excision of a posterior ulcer. (Courtesy of Balfour and Surg., Gynec. and Obst.)

At this stage of the operation, the surgeon is guided by the findings on the posterior wall of the duodenum. If no lesion is found on the posterior wall, the anterior defect is closed in the transverse direction as illustrated in Figure 453. If the posterior ulceration is near the inferior or superior border of the duodenum, the incision is continued around the lumen to include the ulcer. The resulting defect from the excision of this ulcer is then sutured and the anterior defect is closed as was previously done. If the ulcer is centrally located on the posterior wall and at some distance from the pyloric sphincter, Balfour advocates the use of the cautery. After the base of the ulcer has been carefully cleansed by sponging, the point of the cautery is carried around the ulcer until the mucosa is loosened. The cauterization is continued until the ulcer is, to all intents and purposes, excised (Fig. 451). Balfour stressed the point that cauterization be done slowly, in as much

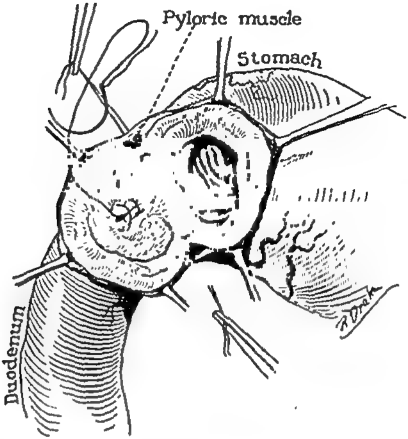


FIG. 452.—Closure with chromic catgut of defect following cautery excision of ulcer. (Courtesy of Balfour and Surg., Gynec. and Obst.)

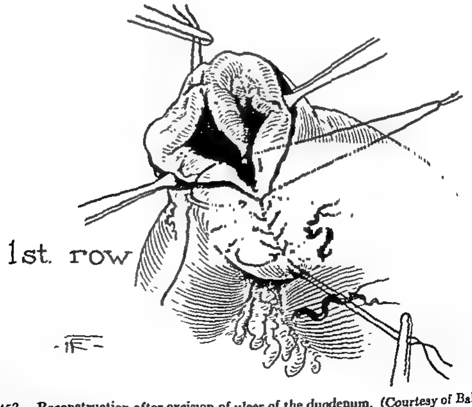


FIG. 453.—Reconstruction after excision of ulcer of the duodenum. (Courtesy of Balfour and Surg., Gynec. and Obst.)

as a low degree of heat is most effective. The defect in the posterior wall is now repaired with a continuous figure-of-eight gastrointestinal catgut suture as illustrated in Figure 452.

If the inspection of the posterior duodenal wall reveals a large ulcer crater, extensive perforation into the pancreas, or multiple lesions excision may not be feasible without unduly constricting the duodenum. In this event, the surgeon should choose some other procedure, and discard the plan of excision.

When the lesion has been dealt with, the anterior defect in the duodenum and pylorus is closed in a transverse direction. As illustrated in Figure 453, the suturing is begun at a point on the lesser curvature side corresponding to the location of the transfixed suture which had been applied at the beginning of the operation. Because of the greater elasticity of the gastric wall, each suture on the gastric side should include enough to match the cut edge of the duodenum. This method of closure is somewhat similar to the closure of a diamond-shape opening in the stomach described in Chapter 54.

QUESTIONNAIRE

1. What conditions will not permit the performance of a posterior gastrojejunostomy?
2. Why should the ligament of Treitz be examined when doing a posterior gastrojejunostomy?
3. If the duodenal-jejunal flexure is acutely angulated, what would you do to correct it?
4. Describe two methods of locating the first loop of jejunum.
5. Give the normal variations in the position of the first loop of jejunum.
6. Discuss the principles in choosing the site of opening of the stomach in posterior [illegible]
7. [illegible] ch contents?
8. [illegible]
9. [illegible] "?
10. [illegible] he stomach in: *a*, antiperistaltic anatomosis; *b*, isoperistaltic anastomosis?
11. On which side of the mid-colic artery is the opening made in the mesocolon? Why?
12. Describe the author's method of delivering the posterior surface of the stomach through the meso-colon.
13. What precaution should be taken when applying clamps to the stomach and bowel?
14. What is the purpose of the clamps?
15. What is a Cushing, Lembert, and Connell stitch?
16. Why is the incision in the jejunum made shorter than that in the stomach?
17. What is the function of the inner row of sutures (Connell)?
18. Why should the mesocolic opening be sutured to the stomach side of the anastomosis?
19. When is an anterior gastrojejunostomy indicated?
20. Describe the technique of an anterior gastrojejunostomy.
21. In radical resection for carcinoma of the stomach what structures are removed?
22. What is the Billroth I operation?
23. Why is it not used in carcinoma of the stomach?
24. Define: Haberer-Finney partial gastroectomy.
25. What is Horsley's modification of the Billroth I?
26. What is the Billroth II method?
27. Discuss its practical value.
28. Give the various modifications of the Polya method of partial gastrectomy.
29. What is the difference between the Balfour and Moynihan modifications?
30. Discuss the importance of searching for metastases in carcinoma of the stomach.

31. Are enlarged lymph nodes a contra-indication to resection? Explain.
32. In resecting ulcers of the stomach, why is a V-shape excision done?
33. Give the technique in V-shape resections of ulcers on the anterior surface of the stomach and at the curvatures.
34. Define the following: vagotomy, vagus neurectomy, vagus resection.
35. Give five different procedures for surgery of the vagus nerve.
36. What is the principle and purpose of the Hollander insulin test?
37. What are the disadvantages of a transthoracic vagotomy?
38. [illegible]
39. [illegible]
40. [illegible]
41. [illegible]
42. [illegible] sophagus.
43. What is the relation of the esophagus to the liver?
44. What is the left triangular ligament?
45. Discuss the anatomy of the vagi according to the findings of Bradley.
46. In what per cent of cases did he find that the vagi could be completely removed trans-abdominally?
47. Describe the technique for subdiaphragmatic vagal neurectomy.
48. How would you mobilize the esophagus?
49. Give the technique of supradiaphragmatic vagal neurectomy.
50. Give the technical principles of the following:
    - *a.* Witzel's gastrostomy.
    - *b.* Soresi's intramural gastrostomy.
    - *c.* Frank's gastrostomy.
    - *d.* Kader's method.
    - *e.* Senn's gastrostomy.
    - *f.* Brunschwig's modification of Senn's method.
    - *g.* Janeway's gastrostomy.
    - *h.* Spivack's modification of Janeway's method.
    - *i.* Beck-Jianu's gastrostomy.
    - *j.* Glassman's double-valve gastrostomy.
51. Describe the technique of Rammstedt's pyloroplasty.
52. Describe the technique of Finney's pyloroplasty.
53. Discuss the value of the Balfour method of excising posterior duodenal ulcers

CHAPTER 58

GALL-BLADDER AND EXTRA HEPATIC BILE DUCTS

By H. A. Oberhelman

GENERAL CONSIDERATIONS

Historical Considerations.—To fully appreciate the present day standards of biliary tract surgery a brief historical sketch of its evolution and unfoldment is quite essential. Since gall-stones were first described by Donatus[5] and Gentile da Foligo[8] in 1348 and their clinical symptomatology first described by Glisson[9] and by Sydenham[32], there is no record of any attempts made for their removal by surgical methods until 1734 when Petit[23] reported the removal of gall-stones from an external biliary fistula and later from an enlarged gall-bladder that had become fixed to the anterior abdominal wall by adhesions. More than a century later Bobbs[2], in 1867, performed the first cholecystectomy in this country on a patient whom he operated upon for what he thought was an ovarian cyst but instead found a huge hydropic gall-bladder containing 50 or more stones. His patient recovered. The real impetus in biliary tract surgery came when such pioneers in surgery as Sims[30] 1878, Tait[33] 1885, and Kocher[19] 1890 began doing cholecystostomies and reporting them in the literature. At the same time Langenbuch[21] performed successfully the first cholecystectomy, although Judd and McIndoe[15] credit Courvoisier[3] with performing the first cholecystectomy a few years earlier. By 1890 Courvoisier was able to report 47 patients upon whom he had done a cholecystectomy. Justus Ohage[27] is given credit for doing the first cholecystectomy in the United States, and his was the ninth reported in the literature.

In so far as surgery of the common bile duct is concerned, Von Winiwarter[24] reported the first cholecystojejunostomy on June 7, 1880 for obstruction of the common bile duct. In 1891, Sprengel[31], going one step farther, reported the first successful choledochoduodenostomy in a patient whose common bile duct he had mistaken for the duodenum at an earlier operation when he had forced a gall-stone from what he thought was the ampulla into the duodenum but in reality had been from the cystic duct into the common bile duct. The late W. J. Mayo[25] in 1905 reported the first successful hepaticoduodenostomy in a patient reported alive eighteen years later.

This work so notably advanced through the efforts of these pioneers has been carried forward by subsequent leaders to the high standard upon which modern biliary tract surgery now rests. Of the many contributions made in this field during the past three decades mention in particular should be made of such men as Mayo-Robson[26] in England, Kehr[18] in Germany and W. J. and C. H. Mayo[24] and Deaver[4] in the United States. It was their inspiring influence that stimulated the younger generation to maintain and even elevate to a higher level the standards governing the procedures in surgery of the biliary tract.

ANATOMY

Embryology.—The liver and the biliary tract are derived from both the mesodermal and entodermal germ layers. They appear as a diverticulum arising in the ventral wall of the primitive foregut at approximately the fourth week of gestation. The proximal portion of this diverticulum differentiates into the liver and hepatic ducts, the distal portions into the gall bladder and cystic duct. The segment between these two portions becomes the common bile duct. The mesenchyme surrounding the foregut provides the fibromuscular and connective tissue of the wall and the entodermal epithelium develops into the lining of the gall-bladder and bile ducts. These structures at first are all hollow but are presently obliterated to form compact cords from rapid cellular proliferation. Shortly small vacuoles form within these cords, which ultimately coalesce and form the lumen of the extra hepatic biliary duct system. In the event that vacuoles fail to appear in the cord-like structures, these cord-like structures remain obliterated and constitute the congenitally obliterated bile ducts in the new born. As to the sphincter of Oddi, little was known, which was equally true of the ampulla of Vater until Schwegler and Boydon[29] in 1937 published their excellent studies. They noted the first signs of the sphincter of Oddi at about the time the embryo reaches a length of approximately 26 mm., when concentric layers of mesenchymal cells make their first appearance at the end of the common bile duct and the pancreatic duct. These cells differentiate into muscle cells concentrically arranged that form a unit entirely independent of the duodenal musculature. Their growth occurs in the direction of the ampulla which gradually pushes into the mucosa producing the eminence that represents the common duct opening into the duodenum, namely, the ampulla of Vater.

ANATOMICAL CONSIDERATION

Gall Bladder.—The gall bladder is pear-shaped 5 to 8 cm. long, having a capacity of 35 to 40 cc. and lies on the under surface of the right lobe of the liver in a shallow depression called the gall-bladder fossa (Fig. 454). It is attached to the liver by some loose connecting tissue traversed by blood and lymph vessels and sometimes by small accessory bile channels. The peritoneum of the liver also known as its capsule is reflected over the entire exposed surface of the gall-bladder. It is conveniently divided into the fundus, body and neck, the neck consisting of a sacculation roughly resembling the letter "S" and forming what is termed, Hartman's pouch (Fig. 455) which may lie close to the common bile duct. Topographically, the fundus of the gall-bladder corresponds to the ninth or tenth costochondral junction in the mid-clavicular line.

Bile Ducts.—The neck of the gall bladder continues proximally as the cystic duct which is 3 to 5 cm. long and 0.3 to 0.5 cm. in diameter and joins with the hepatic duct to form the common bile duct. The epithelial lining of the cystic duct is columnar and thrown up into folds commonly known as the Spiral Valve of Heister (see Fig. 456). It is this structure that makes probing of the cystic duct difficult during surgery. The common hepatic bile duct is formed by the junction of the right and left hepatic duct at the hilus of the liver. It is about 3 cm. long and has an average diameter of 6 mm. This duct as well as the right and left hepatic ducts may vary considerably in length. These ducts lie in the hepaticoduodenal ligament throughout their entire extent.

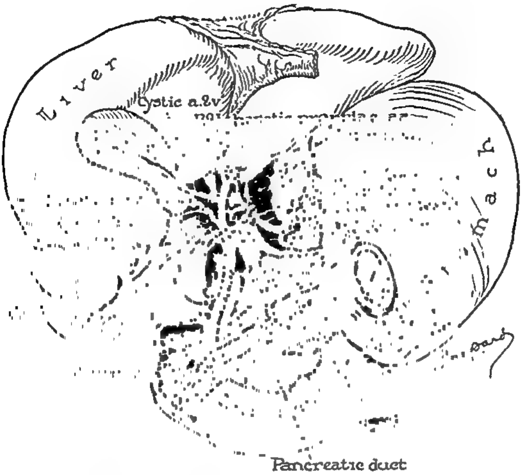


FIG. 454 —An anatomical dissection of the various structures involved in surgical diseases of the biliary tract.

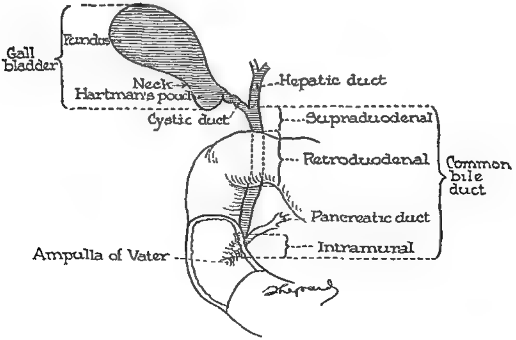


FIG. 455 —Diagrammatic drawing of the five anatomical divisions of the common duct

The common bile duct is formed by the junction of the cystic and common hepatic bile ducts and passes downward behind the pancreas into the second portion of the duodenum. It is 7 to 9 cm. long and 0.5 to 0.7 cm. in diameter except where it enters the wall of the duodenum where it is slightly narrower. It lies in the hepatico-duodenal ligament, to the right of the hepatic artery and in front of the portal vein (Fig. 457). The common bile duct is divided into five segments (Fig. 455) as follows:

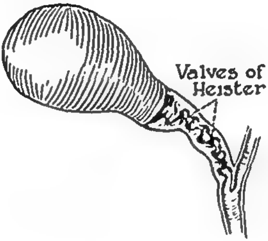


FIG. 456.—The lining of the cystic duct is thrown into folds commonly known as the Spiral Valve of Heister.

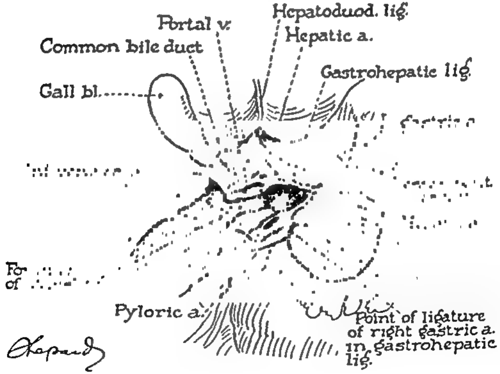


FIG. 457.—Structures anterior to the foramen of Winslow.

1. The supraduodenal segment is 1 to 1.5 cm. long, extends from its beginning down to the upper margin of the first portion of the duodenum and in front of the foramen of Winslow. Just above the upper margin of the duodenum, a small branch of the pancreaticoduodenal artery crosses in front of the duct.

2. The retroduodenal segment lies behind the first portion of the duodenum.

3. The pancreatic segment extends from the inferior margin of the first portion of the duodenum to a point where the duct penetrates the wall of the second portion of the duodenum. Here it lies close to or in a groove of the pancreas, or may even be completely surrounded by pancreatic tissue.

4. The intramural segment is that part within the wall of the second portion of the duodenum.

5. The ampullary segment consists of that part opening into the lumen of the duodenum. This segment is surrounded by both circular and longitudinal muscle fibers that make up the Sphincter of Oddi.

Blood Supply.—The hepatic artery at about the level of the junction of the cystic and common hepatic bile ducts bifurcates into the right and left hepatic arteries which constitute the sole source of blood supply to the liver. The right hepatic artery normally passes behind the common hepatic and cystic bile ducts (Fig. 465, *a*) and at this level the cystic artery arises from the right hepatic artery and accompanies the cystic duct to the neck of the gall-bladder (Fig. 454) where it divides into a deep and a superficial branch. The deep and larger branch penetrates the region between the gall-bladder and the liver, while the superficial and smaller branch supplies the free surface of the gall-bladder.

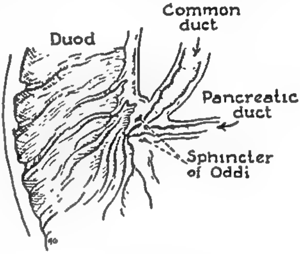


Fig. 458.—Sketch illustrating the sphincter of Oddi.

The Lymphatic Supply.—The ball bladder possesses an elaborate network of lymph channels that drain the submucosal, the fibromuscular and the serosal layers,[4] to empty into one or more lymph glands at the neck of the gall-bladder. Similar lymph channels course through the layers of the cystic and common ducts, as well as the hepatic to converge into the hilar lymph glands.

ANOMALIES OF THE GALL-BLADDER

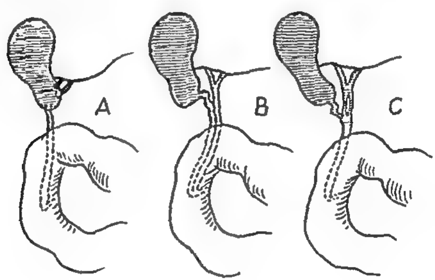
There is probably no other system in the human body that has such frequent anomalies as the biliary system, especially the ducts and arteries. Abnormalities are so frequent that it is difficult to determine at times which is the normal. Flint[7] called attention to this when he made detailed dissections of the biliary system and its blood supply in 200 consecutive autopsies and found that only 34 per cent could be considered as normal. The many variations in both ductal and circulatory systems may be noted on careful study in Figures 465 and 466. Because of their surgical significance, the surgeon must be aware of these anomalies and that therefore great care must always be exercised in routine gall-bladder surgery. Only in this way will he avoid accidents that may be exceedingly grave or even fatal to the patient.

Gall-bladders with congenital anomalies are no more immune to infection and gall-stone formation than those without anomalies. However, anomalies of the

It is of prime importance that these anomalies be recognized when doing a cholecystectomy because the common duct may easily be mistaken for the cystic and be accidentally ligated and either partially or completely excised (Figs. 488 and 489). The cystic duct may be quite long and in general its length will largely depend on what level it meets the common duct (Fig. 461).

When the junction of these two ducts is low, the cystic lies parallel and close to the common hepatic (Fig. 461, *a*), and here again injury to the common hepatic duct may occur in cholecystectomy. Instead of the cystic duct joining the common hepatic duct on the right side as it normally does, it may pass either in front of or behind the common hepatic duct to unite with it on the left side. (See *b* and *c*, Fig. 461.)

Finally the cystic duct may terminate separately in the duodenum, in which event the common hepatic bile duct takes the place of the common duct (Fig. 462, *b*).



ANOMALIES OF THE COMMON HEPATIC DUCT

These may consist of an absence with both hepatic ducts then meeting the common duct at its junction with the cystic; a right accessory hepatic duct joining either the common hepatic duct or the gall-bladder, or one or more small accessory ducts lying in the fossa between the liver and the gall-bladder, and occasionally a duplication. When duplication occurs in the common hepatic duct the lumen of each trunk is small. In the event of acute cholecystitis and cholangitis, jaundice may develop early, because slight swelling in each of the two small channels may result in early obstruction which could not occur in a channel twice the size of either of the two. A few years ago the author encountered just such an example.

The patient had multiple gall-stones with severe cholecystitis and obstructive jaundice. At operation many stones were removed from the gall-bladder and to the author's amazement none could be found in the common duct which was normal in size. The gall-bladder was drained, but no bile ever appeared. Death occurred four days later and at autopsy, the small channels of the duplicated common hepatic duct were completely obliterated by inflammatory swelling, accounting

for the obstructive jaundice. Anomalies of the common bile duct are limited largely to a duplication and to variations in its entrance into the duodenum with respect to the opening of the pancreatic duct (Fig. 463).

When there is an absence of the duct, its failure to form is due to the cystic duct having its own independent junction with the duodenum.

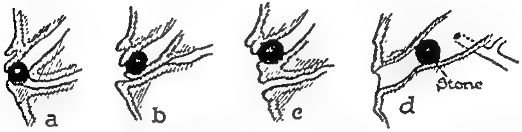


Fig. 463.—Illustrates variations in the terminations of the common bile duct and the pancreatic duct and shows how a stone in the ampulla of Vater may obstruct: (*a*) both common bile duct and pancreatic duct by occlusion; (*b*) common duct by occlusion, pancreatic duct by compression; (*c*) common duct only; (*d*) common duct by occlusion, pancreatic duct by back pressure of bile.

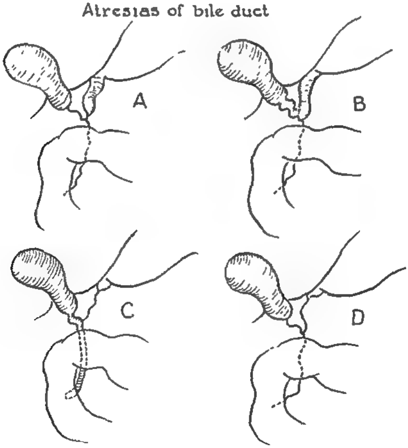


Fig. 464.—Atresias of bile ducts.

CONGENITAL ARTRESIAS OF THE BILE DUCTS

Congenital atresias of the bile ducts are quite rare (Fig. 464). They constitute a serious problem in the newborn because of the technical difficulties involved and the fact that all are poor surgical risks. This subject is discussed more fully under "Reconstruction of the Bile Ducts."

CONGENITAL ANOMALIES OF THE ARTERIES

The arteries that most often cause grief for the surgeon are the hepatic, cystic and gastroduodenal arteries. Their various positions and relationship to each other and to the bile ducts may be noted by a careful study of Figures 465 and 466. Flint's[7] contribution from this standpoint is most noteworthy. He found that the right hepatic artery took its origin from the superior mesenteric artery in 21 per cent of his dissections (Fig. 466, *c*) instead of the main hepatic trunk and in about 5 per cent there was an accessory right hepatic artery (Fig. 465, *c*) usually from the superior mesenteric artery. In most instances the right hepatic artery passes to the right behind the common hepatic duct (Fig. 465, *a*) but in a small minority it

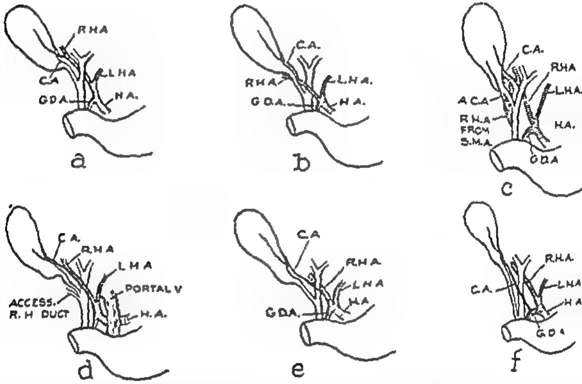


Fig. 465.—Various abnormalities of arteries and ducts in the gall-bladder region (after Flint (C.A.); cystic artery; (G.D.A.) gastroduodenal artery; (H.A.) hepatic artery; (L.H.A.) left hepatic artery; (R.H.A.) right hepatic artery; (S.M.A.) superior mesenteric artery; (A.C.A.) accessory cystic artery; (Portal V.) portal vein; (S P.D.A) superior pancreatico duodenal artery; (Access R.H.) accessory right hepatic duct.

passes in front of it (Fig. 465, *b*). The cystic artery takes its origin from the right hepatic artery in about 90 per cent of instances (Fig. 465, *a*) and its origin in the remaining 10 per cent may be from the gastroduodenal (Fig. 465, *f*) the left hepatic, the superior pancreatico duodenal, or the superior mesenteric. It usually lies above the cystic duct, rarely below, and in a few instances instead of normally passing behind, it may pass to the right in front of the hepatic or common bile ducts (Fig. 465, *e*). *Flint*[22] *found two cystic arteries in approximately 1 out of every* 7 dissections (Fig. 465, *c*) the additional or accessory artery usually lying below the cystic duct and arising from the right hepatic artery or the gastroduodenal. The gastroduodenal artery, encountered in exploring and resecting the retroduodenal segment of the common bile duct, normally lies behind the common duct but according to Eisendrath[6] about 1 in every 5 cases the artery lies anterior to the duct.

CONGENITAL ANOMALIES OF THE ARTERIES

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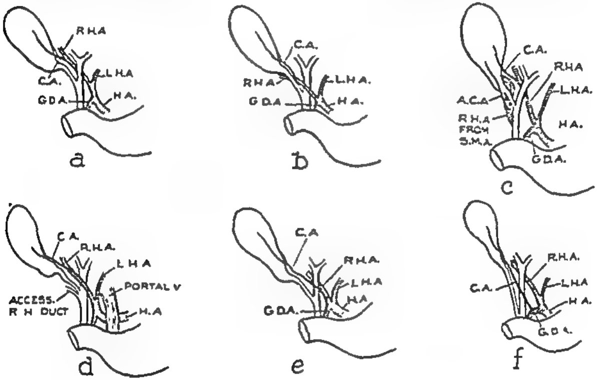

F[illegible]
(C.A. [illegible]
artery [illegible]
cystic [illegible]
R.H.) accessory right hepatic duct.

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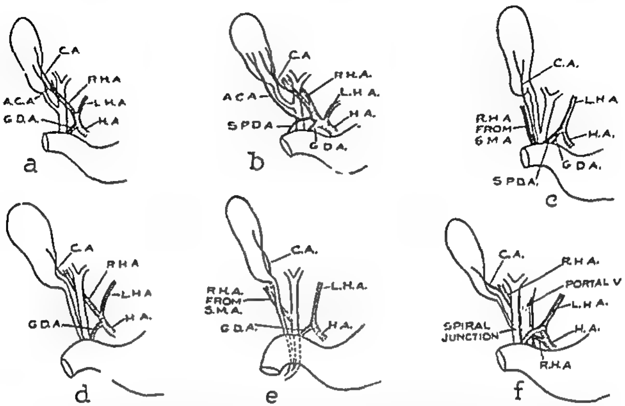

FIG. 166.—Various abnormalities of arteries and ducts in the gall-bladder region (after Flint): (G.D.A.) gastroduodenal artery; (H.A.) hepatic artery; (L.H.A.) left hepatic artery; (R.H.A.) right hepatic artery; (S.M.A.) superior mesenteric artery; (A.C.A.) accessory cystic artery; (Portal V.) portal vein; (S.P.D A.) superior pancreatico duodenal artery; (Acess .R.H. Duct) accessory right hepatic duct

ASSOCIATED PATHOLOGY

When we consider the intelligent surgical treatment of biliary tract disease it is not enough that the surgeon know the pathologic processes involving the biliary tract directly, but it is equally important that he know the associated pathological alterations that accompany such lesions. The presence of gall-stones in the biliary system does not necessarily mean an associated biliary tract disease, but when gall-stones whether they form as the result of infection or from metabolic disturbances, produce obstruction to the flow of bile, then we have the setting created for the development of the associated pathological lesions in biliary tract disease. Infection further augments the gravity of these associated lesions. Contributions to our knowledge of the pathological anatomic alterations involving the biliary tract directly were made by the pioneers in the field of biliary tract surgery. They did not, however, contribute so much to our knowledge of the pathologic alternations affecting contiguous as well as distant organs associated with biliary tract disease. This knowledge is a more recent contribution made notably by MacCarty[22] MacCarty and Jackson[23], Graham and Peterman[10], Heyd and his associates[12] and Judd, Nickel and Wellbrook[16]. Conditions such as chronic pancreatitis, chronic hepatitis and even chronic appendicitis may accompany a simple cholecystitis with cholelithiasis. To what extent duodenal and gastric ulcers are related to cholecystic disease is somewhat speculative, but they frequently occur together. Intestinal obstruction occasionally occurs when a large gall-stone slowly burrows through the wall of the gall-bladder directly into either the duodenum or transverse colon forming either a chlecystoduodenal or cholecystocolic fistula. When such

stones enter the duodenum they lodge either in the upper jejunum or terminal ileum; when they enter the transverse colon they lodge in the ampulla of the rectum and are passed either spontaneously or must be extracted digitally. The relationship of biliary tract disease to chronic arthritis has been stressed by some authors but Judd and Hench[14] state that chronic cholecystitis rarely serves as a focus of infection for chronic infectious arthritis. When a stone becomes impacted in the neck of the gall-bladder or cystic duct, infection commonly sets in producing empyema and gangrene of the gall-bladder leading to either a local or generalized infectious bile peritonitis. Fortunately such an infectious bile peritonitis usually remains localized. Of the associated disturbances that accompany biliary tract disease the most serious occur with obstruction of the common bile duct whether by calculi in the common duct or in the cystic duct at its junction with the common duct, by inflammatory strictures or by neoplasms. In most such instances a chronic hepatitis or a chronic pancreatitis or both are already present, which now are augmented by jaundice and an ascending cholangitis which may become suppurative; the simple pancreatitis may become a suppurative pancreatitis. In either or both of these viscera, multiple abscesses may develop. It is therefore important that the surgeon possess a complete understanding of the far reaching and devastating effects of the associated conditions of biliary tract disease so that he may adequately fortify his patient for surgery.

PATHOLOGIC PHYSIOLOGY

It is not enough that the surgeon know the pathologic anatomic alterations in biliary tract disease, but he must also know equally well the pathologic physiologic alterations that ensue. There is first of all a disturbance of the excretory functions of the liver which is also the last to be restored to normal after the obstruction is relieved. There is definite interference with the digestion of fats and the absorption of the fat-soluble vitamins such as K and B^1 and minerals of which calcium is the most important. The detoxifying function of the liver is usually reduced, which may be recognized by the hippuric acid test of Quick. The plasma proteins likewise show a fall in concentration often leading to a reversal of the albumin-globulin ratio. The ability of the liver to store hemoglobin forming compounds is greatly lessened as shown by the fall in hemoglobin. There is a definite reduction in the synthesis of prothrombin, a significant liver function. The extent to which this function is interfered with may be determined by the Prothrombin Liver Test. When the surgeon recognizes these pathologic physiologic alterations and attempts then to correct them he will have mastered a most important part in the preoperative program upon which so much of the success in biliary tract surgery depends.

The normal function of the gall-bladder is to store and to concentrate bile. The latter is accomplished by the absorption of water. The lining of the gall-bladder contains numerous mucous glands whose function is to secrete mucus. In the event of any obstruction in the cystic duct to prevent the flow of bile in and out of the gall-bladder, mucus continues to be secreted, while the bile pigments are absorbed, leaving a more or less clear fluid known as "white bile." When this condition exists it is known as hydrops of the gall-bladder. The musculature of the gall-bladder has the capacity to contract and empty itself following the ingestion of food particularly of the fatty type. This contraction of the gall-bladder is initiated by a hormone, cholecystokinin isolated by Ivy and Oldberg.[13] Simul-

taneously with the contraction of the gall-bladder the sphincter of Oddi in the ampulla relaxes to permit the flow of bile into the duodenum. During the fasting periods the sphincter of Oddi is in a state of contraction. This forces the bile into the relaxed gall-bladder and at the same time prevents the reflux of duodenal content into the common bile duct. In the state of contraction the sphincter exerts a pressure greater than the secretory pressure of the liver. The not uncommon attacks of so-called gall-stone colic after cholecystectomy is attributed to an increased spasm of the sphincter due to a disturbance of the coördinated mechanism between the gall-bladder and the sphincter resulting in a temporary bile congestion. It is also possible and highly probable that the compensatory dilatation of the common bile duct after cholecystectomy is the result of such spasm.

PRE- AND POST-OPERATIVE CARE

The importance of adequate pre- and post-operative care in biliary tract surgery cannot be too greatly emphasized. The surgeon may have executed his operation with dispatch, with minimal trauma and with faultless technique but if he failed to heed or was ignorant of the significant principles underlying pre-and post-operative care, it might well have been said, "the operation was a success but the patient died." Just as the surgeon recognizes the pathologic anatomic alterations in biliary tract disease, so he also recognizes the pathologic physiologic alterations in this disease, and is fully aware of the fact that the former cannot be successfully treated without also treating the latter. The successful treatment of the former constitutes an earlier contribution made principally by surgeons, while the successful treatment of the latter is a contribution made more recently by physiologists.

In the pre-operative management it is assumed that the patient has been carefully studied and a diagnosis of biliary tract disease is fully warranted for which surgery is the treatment indicated.

In general, surgery of the biliary tract is for the most part elective and only rarely emergency. In the uncomplicated group of patients, whether the diagnosis is chronic calculous cholecystitis or chronic non-calculous cholecystitis, the pre-operative program is considerably more simple than for the complicated group. It is common practice for the uncomplicated patient to be admitted to the hospital the day before surgery and have only the routine blood work and urine analysis done, and be given a light evening meal and a mild sedative for sleep. On the following morning a cleansing enema is given, breakfast is withheld, and a hypodermic of morphine sulphate gr. $\frac{1}{6}$ and of atropine sulphate gr. $\frac{1}{150}$ is administered one hour before surgery. Before going to the operating room a Levine tube is passed through the nose into the stomach to be left in during surgery and for several days post-operatively. It keeps the somach collapsed during surgery providing much more room in the operative field than would otherwise be provided. This tube furthermore prevents any vomiting, thus making it impossible for any vomitus to be aspirated into the trachea and bronchi which may easily occur when this precaution is not taken.

While the above routine may be adequate in most instances, the author feels that certain additional pre-operative measures are essential in conducting the patient safely through an operation on the biliary tract.

Since the liver plays such a major role in maintaining body metabolism, and since the biliary system and the liver constitute a common anatomic and physio-

with the glucose in normal saline. The saline is needed to prevent depletion of chlorides by the continuous suction. Vitamin C—Solu-B, and penicillin may be given with the intravenous fluids. Hourly blood pressure and pulse records should be made during the first twenty-four hours. Adequate sedatives are essential for the first forty-eight hours or longer if necessary. Deep breathing exercises should be encouraged and frequent change of position, with the patient helping himself as much as possible. Early ambulation is highly desirable even mandatory on the second post-operative day. Vitamin K should not be used post-operatively unless the prothrombin is down because it increases the coagulability of the blood and thus augments the tendency to thrombophlebitis or phlebothrombosis. On the third day the Levine tube may be withdrawn, fluids taken by mouth, and a soft diet as tolerated. The cigarette drain which has been shortened on the first post-operative day may now be entirely removed. The remainder of the post-operative course should be uneventful.

In the complicated group of patients, additional measures are necessary in addition to those just mentioned for the uncomplicated group. In the first place oxygen by nasal catheter may be necessary, and when used should not be used too long because it allows shallow breathing which means incomplete inflation of the pulmonary alveoli. Furthermore, the administration of intravenous fluids of 5 per cent glucose, plasma or blood started in the operating room must be maintained. The rubber tube for the drainage of the gall-bladder or common duct should be connected with a bottle at the bedside. If the tube is in the gall-bladder it may be removed in about ten days; if in the common bile duct not for some time. If the loss of bile is prolonged for reasons that cannot be corrected for some time, Judd and White[17] recommend that the bile be collected and refed to the patient by stomach tube, supplemented with generous amounts of bile salts. Vitamin K should be continued especially for the jaundiced patient to aid in the formation of prothrombin and thus prevent bleeding. Even though there seems to be no need for blood transfusions from the standpoint of red blood cell count and hemoglobin readings, yet Adams and associates[34] noted by making daily determinations of the blood serum protein a progressive fall in blood proteins starting on the third post-operative day as a rule. They also noted that a protein deficiency did not manifest itself clinically until the sixth or seventh post-operative day. As a consequence, *they recommended blood transfusions routinely on the third or fourth post-operative day* when the proteins begin to fall rather than wait until they have become clinically deficient. In the event no blood is available, plasma may be substituted.

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CHAPTER 59

GALL-BLADDER AND EXTRA HEPATIC BILE DUCTS

By H. A. Oberhelman

SURGICAL PROCEDURES

Anesthesia.—In surgery of the biliary tract it is of major importance that a suitable anesthetic be selected. The large majority of gall-bladder patients are past or nearly past middle life and many of these in the upper age bracket and therefore not always the best surgical risks. Furthermore, there is frequently already considerable hepatic damage and an all out effort must be made to select an anesthetic with the minimum risk. Such a selection may readily be made in clinics where expert anesthetists are always available, so that little if any responsibility need fall upon the operating surgeon. This, unfortunately is not true in the average community hospital the country over, where after all more gall-bladder surgery is done than in highly specialized clinics and hospitals. Prolonged or too deep anesthesia in surgery of the upper abdomen is not infrequently conducive to pulmonary complications. When this occurs it is not the fault of the anesthetic, but may be the fault of the anesthetist. The question might be asked "What anesthetic or combination of anesthetics constitutes the anesthetic of choice in biliary tract surgery?" This question may be answered from two viewpoints and both answers could be correct. One answer could be the anesthetic having the least toxic effect upon the liver; the other answer could be that it is the anesthetist best trained in to administer. This seems to justify the statement not infrequently made, namely, "It is not what anesthetic but *who is the anesthetist.*" Therefore, it is important that all anesthetists be well trained not only in selecting the best anesthetic for the individual patient but be equally well trained in its proper administration.

The following anesthetics or combination of anesthetics have been found useful in the author's experience:

1. Novocain 0.5 to 1 per cent for local infiltration or block anesthesia. It is probably most useful in poor surgical risks where only the abdominal wall needs to be opened for draining a badly infected and distended gall-bladder.

2. Spinal anesthesia has proven useful and is the anesthetic of choice by some surgeons.

3. Pentathol sodium, 2 per cent intravenously has proven itself useful, mainly, however, as a means of induction for inhalation anesthesia, but may occasionally provide adequate relaxation alone.

4. Inhalation anesthesia, such as nitrous oxide, ethylene or cyclopropane in combination with oxygen, carbon dioxide or small amounts of ether is excellent. Ethylene or cyclopropane supplemented guardedly with curare (Introcostrin, Squibbs) 3 cc. intravenously has proven quite popular.

5. Ether, alone or preceded by gas induction, is in the opinion of the author the safest anesthetic in the hands of the greatest number administering anesthesia the

country over. That is why the author frequently has the least misgivings when the only anesthetic in sight is a can of ether, and an anesthetist who knows how to administer it.

Position on the Table.—The standard position of the patient on the table is in the supine. In the earlier days when cholecystectomy was replacing cholecystostomy, and surgery of the bile ducts was being established, the patient's position on the operating table was considerably emphasized. It was found that when the trunk was elevated at the level of the costal margin the liver would be lifted forward, exposing the subhepatic space and therefore making the gall-bladder and bile ducts more readily accessible. This was accomplished by placing sand bags or an air cushion under the patient's back, or by so-called "breaking of the table" at the desired level or by placing the gall-bladder lift, equipped with a crank, which when turned would raise the patient's back to any desired level. While this position may have aided somewhat in easing the approach to the biliary tract, other more humane measures have been substituted so that most surgeons, including the author, have not only abandoned the "lift" as an aid but even discourage its use. In fact, the same elevating effect is accomplished even more effectively by placing traction upon the upper end of the divided suspensory ligament which lifts only the liver and leaves the patient's spine in the normal position.

Incision.—In selecting an incision for the approach to the gall-bladder and bile ducts, it should be one having the following features:

1. Easily made and easily closed, giving dispatch to this part of the operation.
2. Preservation of the nerve and blood supply to the rectus abdominalis muscle to prevent incisional hernias.
3. Provide adequate exposure for thorough exploration of the bile passages and to facilitate the performance of the necessary surgical procedures without undue hazard.
4. An incision that can easily be lengthened with the least delay.

The following incisions for the most part possess the above mentioned qualification:

1. The vertical right upper para-median incision extending from the costal margin down to below the level of the navel. The anterior rectus sheath is divided vertically near the midline, the rectus abdominalis muscle retracted laterally and the posterior sheath and peritoneum divided likewise in the same direction. This incision has many adherents.

2. The transverse incision recommended by Gurd is preferred by a considerable number of surgeons. This incision is carried transversely from the tip of the right twelfth rib to a point 2 or 3 cm. to the left of the midline and approximately 2 to 3 cm. above the level of the navel. Division of the anterior rectus sheath, the external and internal oblique is likewise made in the transverse plane. The rectus abdominis muscle may either be retracted medially or divided if greater exposure is required; the posterior rectus sheath, transversalis muscle and peritoneum are then divided in the transverse direction.

3. The so-called high right rectus incision used and advocated by the Mayo Clinic is the author's choice of gall-bladder incisions (Fig. 467). This incision is made from a point just to the right of the tip of the ensiform cartilage, extending obliquely downward and to the right to below the level of the navel. The anterior rectus sheath is divided in the same line, the rectus abdominis muscle is split parallel with its fibers and then the posterior rectus sheath and peritoneum are divided in

line with the split rectus muscle. This incision affords satisfactory exposure of the gall-bladder and bile ducts and can easily be extended downward to expedite any additional surgery in the right lower quadrant.

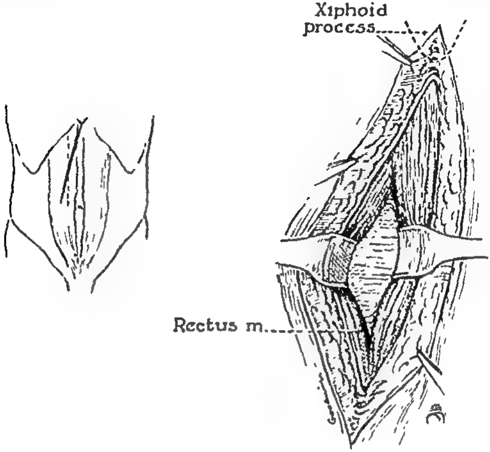

FIG 467.—Rectus incision.

CHOLECYSTOSTOMY

In the latter part of the nineenth century when the surgical treatment of biliary tract disease was becoming established, cholecystostomy (drainage of the gall-bladder) was the operation of choice. It was and is a relatively safe procedure and is a convenient method for the occasional operator. However, surgeons soon learned that many of their cholecystostomized patients developed recurrent symptoms of gall-stone colic. This prompted the more radical procedure of removing the gall-bladder which has resulted in a lower incidence of recurrence. While cholecystectomy is now the operation of choice and has therefore largely replaced cholecystostomy, there are still certain limited conditions in which the latter is and should be the operation of choice. It is the authors impression that too often unnecessary risks are taken by surgeons in doing a cholecystectomy when a cholecystostomy should have been done instead. It need not be a question of lack of technical ability but a question of proper surgical judgement. The conditions for which cholecystostomy is indicated are: (1) Elderly people who are poor surgical risks and whose life expectancy is short, will do much better with cholecystostomy than with cholecystectomy. (2) Extreme obesity, especially when the patient is acutely ill or when the physical hazards make accessibility difficult. (3) Severe inflammatory conditions such as empyema, gangrene and threatened perforation of the

gall-bladder wall. In these conditions the gall bladder is large—often lying against the anterior abdominal wall. (4) Severe inflammation of the common bile duct with ulceration from stones associated with stones in the gall-bladder. As healing and fibrosis ensues a benign stricture of the common bile duct may develop and if the gall-bladder is still available and not too badly diseased it will be a most convenient structure for a by-pass operation. (5) Patients who already have some major disease and develop gall-stones have a much better chance for recovery with drainage than with removal.

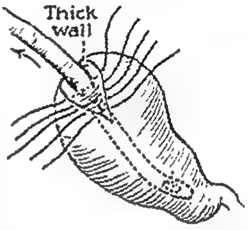

FIG. 468.—Alternate method of cholecystostomy when gall-bladder wall is thickened.

Technique.—The type of incision may be one of several depending in part on the fancy of the surgeon but more on the severity of the inflammatory process or on the other conditions just mentioned, requiring surgical intervention. *In the event of an empyema or gangrene or both of the gall-bladder, a short muscle splitting incision should be made over the most prominent part of the palpable gall-bladder area.* This incision need be only long enough to just expose the fundus of the gall-bladder to insert a rubber tube for drainage. Under no circumstances should the dissection be carried beyond this limited field for it will only break down the effective barrier nature already has placed around the gall-bladder. An aspirating needle should first be introduced into the distended gall-bladder and decompressed. It *should then be incised long enough to permit the introduction of a gall-stone scoop* for the removal of the gall-stones. In the event of an impacted stone in the neck of the gall-bladder or in the cystic duct that cannot be dislodged with the gall-stone scoop, *and if the patient is not too ill, it is permissible to pass the hand palm side up along the under surface of the gall-bladder breaking the plastic adhesions enough to allow digital pressure upon the duct to dislodge the impacted stone.* If the patient is too critically ill to permit this degree of manipulation the stone may be left alone. It may subsequently be expelled through the drainage tube, and if not a second operation will be necessary at a time when the surgical risk will be much less. If the gall-bladder wall is too thick or too friable to permit the use of the usual purse-string suture, *the tube may be held in the gall-bladder by closing the opening in the fundus with interrupted sutures (Fig. 468), with the last suture also passing through the rubber drainage tube.*

In the event the condition for which drainage is indicated permits free incision into the peritoneal cavity, the abdomen is first explored gently and no viscera should be permitted to escape through the incision while doing the routine exploration. In the event direct inspection is necessary in addition to manual palpation, the gall-bladder, if mobile enough may be delivered into the operating field; if not the surrounding viscera may be "packed-off" by warm (not hot) moist laparotomy packs to allow direct intra-abdominal inspection. By such means the subhepatic space is exposed. If this space is not obliterated by fibrous adhesions the index and middle fingers of the left hand may be passed through the foramen of Winslow and with the aid of the left thumb the structures in the hepaticoduodenal ligament may easily be palpated such as the common bile duct, portal vein, hepatic artery and periportal lymph glands. The hepatic duct, pancreas, duodenum and the liver

are inspected. If stones occur in the cystic duct, a reasonable attempt should be made to dislodge them upward into the gall-bladder, or if they occur in the ampulla, to dislodge them into the duodenum. Should this not be feasible then an adequate incision must be made through the duct over the stone under direct vision and then expressed. (See technique for Choledochoduodenostomy.) In the event the sub-

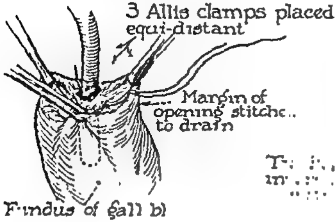

FIG. 469

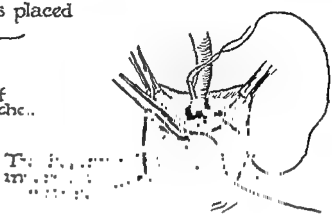

FIG. 470

FIG. 470.—The second stitch is shown passing through the gall-bladder wall only.

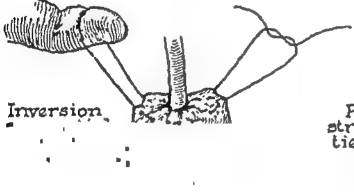

FIG. 471

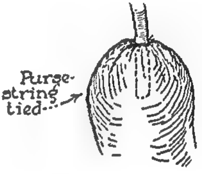

FIG. 472

FIG. 472.—The purse-string suture has been drawn snugly around the tube, further invaginating the fundus and completing the operation.

hepatic space is obliterated by fibrous adhesions, and stones are found in the gall-bladder, just enough of the adhesions are divided to expose the fundus and allow three Allis clamps to be applied *equi-distantly to the fundus* for control of the viscus. The fundus is then opened and the stones are removed with a scoop or forceps. If there are many small concretions irrigation with sterile water may aid in their removal. *However, in the author's experience the best method to remove* such small concretions is to take the open end of a gauze sponge, carry it far down into the neck with a forceps, then firmly packing the entire sponge into the gall-

bladder. As the sponge is then slowly withdrawn, the small concretions will be seen caught in the meshes of the gauze. This is repeated several times until all are removed. When this is completed a medium sized rubber tube (Figs. 469 to 472) is introduced into the gall-bladder for 3 to 4 cm. With the gall-bladder held by Allis clamps the rubber tube is tied to the gall-bladder wall by a single interrupted No. 0 chromic catgut suture. This same suture is then used as a continuous Lembert around the entire tube, biting through the edge of the gall-bladder wall only, except at the halfway mark where the bite passes through both tube and gall-bladder wall. The two ends are then tied while the assistant invaginates the edges of the wall about the rubber tube. This brings tube and gall-bladder wall snugly together. To provide additional precautions against leakage of bile a second purse-string suture (Fig. 471) of No. 0 chromic catgut is placed in the fundus wall 0.5 to 1 cm. out from the edges completely around the tube. At the half-way mark a loop of catgut is left loosely so that when upward traction is made on this loop and the two free ends, the wall of the fundus is further invaginated by pushing down on the rubber tube. As traction is maintained on the free ends the loose loop is taken up and the ends then snugly tied. This completes the procedure. A cigarette drain should be placed in the subhepatic space through a stab opening at the costal margin in the midaxillary line. The rubber drain should be brought out through the abdominal wall in a line that is in keeping with the direction of the long axis of the gall-bladder. This will not only avoid bending and kinking of the tube and thereby interfere with free bile drainage, but also permits the gall-bladder to resume its normal position adding much to the patient's comfort. The peritoneum is closed with continuous No. 1 plain catgut suture, the rectus sheath with No. 1 chromic catgut as a continuous plus several interrupted sutures, and the skin with continuous lock or interrupted vertically placed mattress sutures of black silk. The cigarette drain may be removed partly in twenty-four hours and completely in forty-eigth hours. The rubber tube will remain in the gall-bladder for two weeks or as long as is necessary for the chromic catgut to absorb.

Result.—The immediate result after cholecystostomy is as a rule most gratifying because the stones causing the pain have been removed. The sometimes discouraging feature is the recurrence of stones that require a second operation. This is much less true following cholecystectomy. According to Cole, as high as 50 per cent of patients with cholecystostomy will have a return of symptoms within four years. This is considerably higher than what has been the author's experience. The mortality rate, strange as it may seem, for patients with cholecystostomy is higher than with cholecystectomy. This, however, is not strange because the patients with cholecystostomy are usually far more critically ill and much poorer surgical risks than patients having an elective cholecystectomy.

CHOLECYSTECTOMY

Indications.—As already indicated, cholecystectomy has to a large extent replaced the earlier operation of cholecystostomy, and has now for many years been the operation of choice. No field of surgery presents greater difficulties than the field of biliary tract surgery. Lord Moynihan has put it so appropriately when he stated: "Surgery of the gall-bladder is difficult, extremely difficult. I hold it to be of greater technical difficulty and to present more problems for immediate accuracy in judgment than any other branch in surgery. If I might presume to offer

any advice to the surgeon who has not had great and continued opportunities for practical work, I would suggest to him to leave the surgery of the gall-bladder alone."

In general, it might be stated that the indications for cholecystectomy are the contra-indications for cholecystostomy in cholecystic disease. These indications are as follows:

1. **Chronic Non-calculus Cholecystitis.**—This condition constitutes one of the real problems in diagnosis. Usually three types are met with, one a chronic fibrous cholecystitis, another a cholesterosis or commonly designated as "strawberry" gall-bladder, and the third the chronic catarrhal cholecystitis. These conditions cause indefinite upper abdominal distress associated with belching and more or less intolerance of fried and fatty foods. The diagnosis is more often arrived at by a process of elimination than through positive clinical and laboratory findings. The three forms cannot be differentiated pre-operatively. Surgery in these conditions is advised only after a reasonable period of medical management fails to give these patients relief.

2. **Chronic Cholecystitis with Calculi.**—It is to this group that most of the patients with cholecystic disease belong. They fall into three fairly well defined types:

1. Those patients having an occasional to frequent attacks of gall-stone colic of short duration.
2. Those having a mild upper abdominal distress associated with belching and intolerance of fatty foods.
3. Those who are entirely symptomless, and in whom gall-stones were disclosed as an incidental finding in a routine x-ray examination.

The patients of the first and second types belong to that group whose surgery is of the "elective" variety. Those patients comprising the third type face either one of two alternatives. One is for the patient to wait until positive symptoms appear and then do surgery. The other is to have the gall-bladder and stones removed as a prophylactic measure. More will choose the former alternative. In general, early surgery should be advised to avoid serious complications that may follow when surgery is delayed too long.

3. **Acute Cholecystitis With or Without Stones.**—In the last decade much emphasis has been placed upon early surgery in this condition instead of following the long established method of conservative treatment followed by surgery later. In these patients, instead of the attacks of gall-stone colic being transitory, they have become sustained, indicating that the stone has become impacted in the neck of the gall-bladder or in the cystic duct. If there is no relief after twenty-four to forty-eight hours, surgery should be done promptly before gangrene develops or perforation occurs. Fortunately only a small minority of patients fall into this group, and therefore this problem of emergency surgery does not come up too frequently. Should the condition not come under observation until the fourth or fifth day after onset, and it is certain that the condition is subsiding, then surgery should be delayed. If there are no signs of abeyance, and gangrene and perforation of the gall-bladder are imminent, in the author's opinion, cholecystostomy is the operation of choice. Not infrequently under such pathologic conditions the gall-bladder, after healing and organization have taken place, consists of a small contracted mass of tissue, either with partial or complete obliteration of the gall-bladder

lumen. In the event a stone becomes impacted in the cystic duct, with little or no infection of the gall-bladder a mucocele or hydrops (white bile) of the gall-bladder develops. This constitutes a definite indication for cholecystectomy.

4. **Trauma to the Gall-Bladder.**—Trauma might constitute an indication for cholecystectomy depending upon the location of the injury. If it should involve the fundus, simple cholecystostomy or even simple closure of the opening would suffice, whereas if the neck were involved, cholecystectomy would be indicated.

5. **Tumors of the Gall-Bladder.**—Of the benign tumors, papillomas and polyps occur but are rare. They are seldom diagnosed pre-operatively and when found at operations, cholecystectomy should be performed. Of the malignant tumors, carcinomas account for most of them, while sarcomas rarely occur. The incidence of carcinoma is less now than fifteen years ago, presumably because many gall-bladders formerly cholecystostomized are now cholecystectomized, and the fact that gall-stones occur with carcinoma of the gall-bladder in 80 to 90 per cent of instances, no doubt some are removed before carcinoma develops or has become grossly recognized. Carcinoma *per se* is often not diagnosed pre-operatively and in its early stages may represent an incidental finding. In the author's experience, many patients with carcinoma of the gall-bladder are inoperable when they come in for treatment.

SURGICAL TECHNIQUE FOR CHOLECYSTECTOMY

The incision may be a right upper paramedian, a transverse, a subcotal or a high right rectus muscle splitting. The author prefers the last mentioned incision. When the peritoneum is opened the left hand is passed into the peritoneal cavity to first explore the lower half of the abdomen and the left upper quadrant. These regions consist of the unexposed portion of the abdomen and unless some unusual pathology is found, exploration by palpation only will suffice. The structures in the right upper quadrant are now inspected and palpated, such as the stomach, duodenum, pancreas, liver, transverse colon and biliary tract. This, the author finds, can best be done with the surgeon standing on the left side of the patient. This position permits the surgeon to pass his left hand free and easy into the right upper quadrant. This keeps the hand, wrist and forearm relaxed, which is very essential in providing the maximum delicacy of touch to the fingers. The left hand is now passed first over the right lobe of the liver, then under it to palpate the structures in the hepatico-duodenal ligament. This is done by passing the left index and middle fingers through the foramen of Winslow into the lesser peritoneal cavity and then with the aid of the left thumb, the common bile duct, the portal vein and lymph glands may be carefully palpated. It will be noted that all during this phase of the operation the left hand, wrist and lower forearm have served as an effective laparotomy pad in retaining the adjacent vicera within the peritoneal cavity. The surgeon now returns to the right side of the patient.

To facilitate good exposure of the gall-bladder a curved artery forceps is applied to the fundus and traction made upward. At the same time the suspensory ligament is divided between forceps and upward traction made on the upper forceps, rolling the liver forward and upward (Fig. 473).

Laparotomy packs are used in retaining the adjacent viscera. With the gall-bladder now adequately exposed, one may proceed to remove it in one of two ways, either by starting at the cystic duct and dissecting upward to the fundus, or starting

at the fundus and dissecting downward to the cystic duct. If the surgeon chooses the former method, he will maintain upward traction on the forceps already applied to the fundus, and place a second forceps on the neck through a small peritoneal incision made at this level (Fig. 474). By blunt dissection the cystic duct is isolated at its junction with the neck of the gall-bladder (Fig. 474), doubly clamped, and each clamp ligated. The stump of the cystic duct may be doubly ligated to prevent leakage of bile. The cystic artery is next identified, doubly ligated and divided

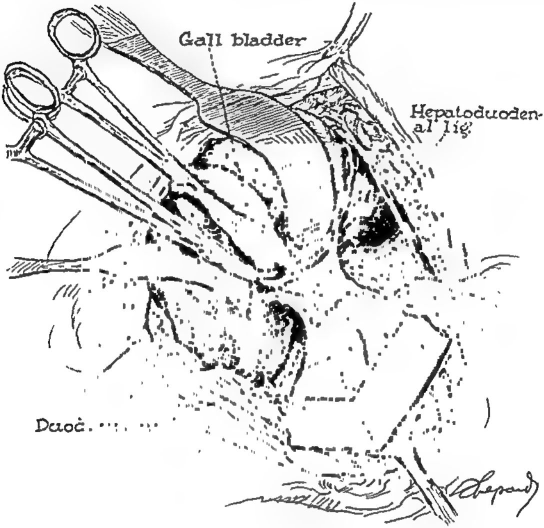

FIG. 473 —Cholecystectomy. Forceps applied on fundus and Hartmann's pouch.

between ligatures. It is important that the forceps on the proximal end of the cystic artery be removed at once by ligating the artery and thus avoid tearing it off accidentally and causing serious hemorrhage. By sharp dissection, the peritoneal attachment to the liver is now divided, leaving an adequate margin on each side and at the fundus to permit peritonealization of the exposed fossa after the gall-bladder is removed (Fig. 475). In carrying out this phase of the operation, the surgeon must proceed cautiously and be constantly on the alert for any anomalies whether arterial or ductal. If any accessory ducts are encountered between the gall-bladder and the liver, they should be individually ligated to prevent unnecessary bile drainage. It is paramount that injury to the hepatic artery be avoided and that no structure be divided unless certain of its identity.

When the gall-bladder is removed from the fundus down to the cystic duct, the fundus is likewise grasped with a curved forceps and upward traction made. With a sharp knife, a transverse incision about 2 cm. long is made through the peritoneum over the fundus about 1.5 cm. from its attachment to the liver (Fig. 476, *a*). The incision already made is now carried down through the peritoneum with scissors

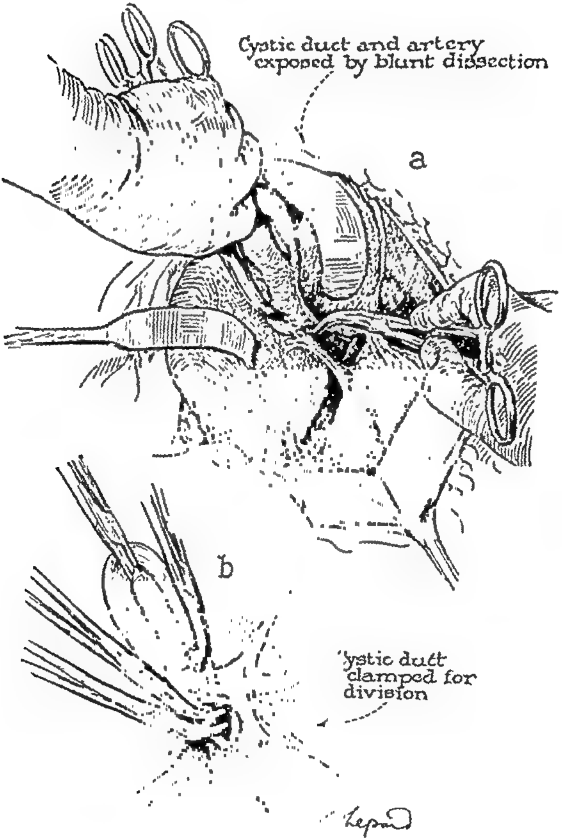

FIG. 474.—(*a*) The cystic duct and cystic artery are being dissected free; (*b*) the cystic duct is doubly clamped with Moynihan forceps.

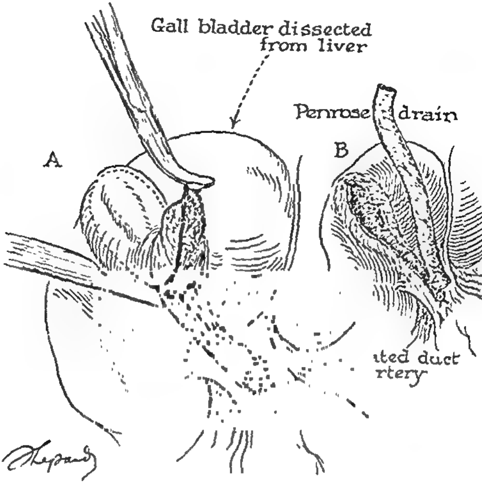

Fig. 475.—Shows method of freeing the gall-bladder by sharp dissection from the liver bed.

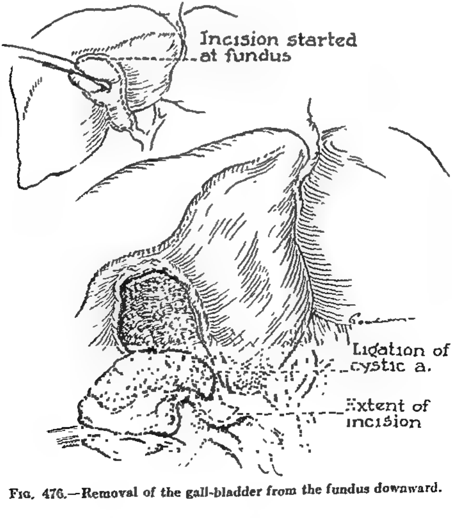

Fig. 476.—Removal of the gall-bladder from the fundus downward.

on each side of the gall-bladder to the cystic duct. Here the two incisions meet over the under side of the cystic duct (Fig. 477). By blunt dissection the gall-bladder is now separated from the liver, using for the most part a gauze sponge over the fingers and peeling the liver away to where the cystic artery passes onto the gall-bladder, which is about two-thirds of the way down to the cystic duct. By blunt dissection the cystic artery is isolated, clamped, cut and immediately ligated with No. 1 chromic catgut. Occasionally the cystic artery lies on the under-side of the gall-bladder. With all the structures but the cystic duct divided, any stones in the gall-bladder may now be milked upward into the fundus. A gall-bladder clamp is now applied to the cystic duct, being certain that the common and

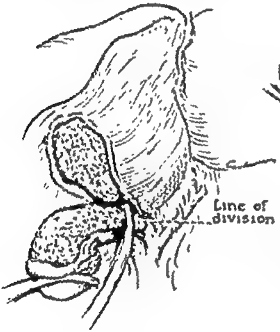

FIG. 477

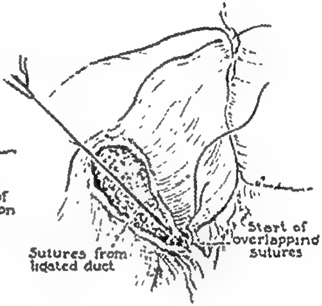

FIG. 478

FIG. 477.—Shows dissection down to where cystic artery enters gall-bladder.
FIG. 478.—Stump of cystic duct buried in gall-bladder fossa.

hepatic ducts are not included in the bite (Figs. 477 and 478). With No. 1 chromic catgut the cystic duct is ligated by transfixion, and while the ligature is being drawn tight the jaws of the clamp are opened just enough to allow the slack to be taken up securely and then shifted upward for 1 cm. and again closed. The cystic duct is then divided distal to the ligature. The ligature is left long so that the stump of the cystic duct may be drawn upward and buried in the gall-bladder fossa as the edges are closed over the fossa for peritonealization and control of bleeding from the cut edges of the capsule (Fig. 479).

The field is now carefully inspected for any bleeding and if none is noted, a Penrose cigarette drain is placed into the subhepatic space. This, in the opinion of the author, is most urgent. It may be introduced through a stab opening on the side or through the incision itself. Leakage of bile may occur from overlooked and divided accessory bile channels or from the stump of the cystic duct caused by a slipped ligature or from pressure necrosis of the duct by the ligature before healing takes place. This drain will prevent a serious bile peritonitis. It is removed in from forty-eight to seventy-two hours, although some surgeons do not remove the drain before the eighth day. Closure of the incision is made in layers, using catgut

silk or cotton sutures for the peritoneum and rectus sheath. Silk or metal clips are suitable for the skin.

To briefly compare the two procedures of cholecystectomy, the author favors the method from the fundus to the cystic duct. There is no doubt more bleeding than what occurs by dissecting from the cystic duct to the fundus, and also one is possibly more apt to perforate the gall-bladder and spill its content in a clean operating field. However, as a rule the bleeding is negligible and any spilling is very promptly removed by the aspirating tip. On the other hand there are several distinct advantages by going from the fundus to the cystic duct.

1. It is far easier to find the line of cleavage at the fundus than at the neck where the dissection is carried directly to the trunks of the arteries and ducts instead of to their terminations.

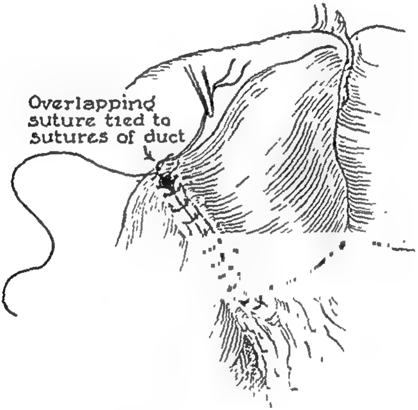

FIG. 479.– Shows entire gall-bladder fossa peritonealized by a running suture.

2. Dissecting down from a known landmark as the fundus gives the operator a clearer view of the anatomic relationship as one approaches the cystic artery and cystic duct.

3. When the gall-bladder is buried in adhesions there can scarcely be any other approach, but to begin at the fundus.

4. Many gall-bladders are removed of necessity by the occasional operator or even by the inexperienced operator and for such individuals the fundal approach would be the safer. An interesting observation in this connection is related by Allen who had occasion to evaluate both methods. Several years ago he reviewed all the case histories of patients entering the Massachusetts General Hospital with operative injuries to the bile ducts. In every instance but one he was able to determine that the gall-bladder had been removed from below upward. He and his staff then adopted the method of removing the gall-bladder from the fundus to the

duct. During a six year period that the operations were carried from the fundus to the duct, they had no ductal injuries.

Results.—By and large the end results of cholecystectomy may be considered good. The best results are obtained in those patients who have had gall-stones while the less satisfactory results are obtained in those patients in whom a non-calculous cholecystitis was found.

CHOLEDOCHOTOMY AND CHOLEDOCHOSTOMY

Surgical procedures on the bile ducts which earlier were thought to be impossible are now relatively common. Practically the only pathologic condition remaining so far for which surgery has not provided relief are certain congenital atresias of the bile ducts and the inoperable carcinomas. The success which has been met

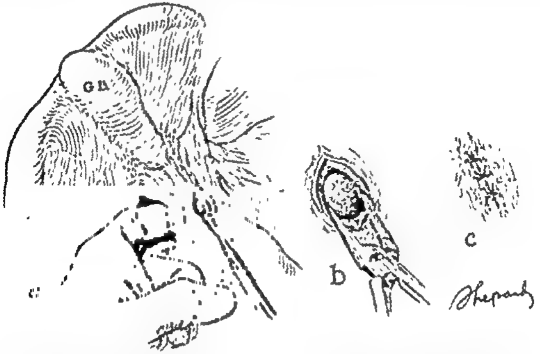

Fig. 480.—Choledochotomy for removal of stone: (*a*) shows method of supporting the duct with a finger in the foramen of Winslow; (*b*) shows stone being extracted; (*c*) shows repair of the incision in the duct, without drainage.

with in surgery of the remaining conditions of the bile ducts has provided wide experience for a large number of surgeons throughout the country so that there is now no longer any hesitation in attempting such surgery.

Indications for Choledochotomy and Choledochostomy.—The indications for choledochotomy or choledochostomy, in the author's opinion, should be restricted to certain definite conditions. To advocate exploration of the common bile duct in routine cholecystectomy as advocated by some surgeons should be definitely discouraged. In the absence of jaundice or any history of jaundice and in the presence of a normal sized common bile duct, the common bile duct should be left alone. In recent years strictures of the common bile duct appear to have increased. This may well be attributed, in part at least, to published writings in which such a routine exploration is advised. On the other hand one must be aware of the various indications that do occur for such exploration. The indications for opening the common bile duct are as follows:

1. The presence of palpable stones. There may be one or many, and in addition to being in the common duct there may be one impacted in the ampulla of Vater or in the hepatic ducts. When a stone is impacted in the ampulla, there is usually severe jaundice, although the author has removed small stones from the ampulla that have caused no jaundice whatever and would permit the passing of a 4 mm. probe. However, the duct is usually dilated and may be so greatly dilated to be mistaken for the duodenum.

2. The common duct should be explored in the presence of jaundice or a history of jaundice. It is the author's experience that under such conditions the common duct is invariably dilated to a lesser or greater extent. Obstruction of the intermittent type will produce a much greater dilatation than obstruction of the sustained type.

3. The presence of a dilated and thickened common bile duct without jaundice should be carefully investigated because the obstruction may be only partial, due to a small stone, a chronic inflammatory stricture or a chronic pancreatitis.

4. In the presence of sustained jaundice of the obstructive type resulting from benign or malignant tumor of the ducts, the ampulla of Vater or head of the pancreas, exploration of the duct is indicated.

Choledochotomy and Choledochostomy.—To undertake any type of common or hepatic duct surgery requires familiarity with their normal as well as abnormal anatomy. It is equally important that the surgeon have a thorough anatomic knowledge of all the adjacent structures. The same incision and the same technique are followed as already described for inspecting the biliary ducts preliminary to cholecystectomy. If adhesions are present from previous inflammatory processes or from previous surgery, they are carefully divided either bluntly or by sharp dissection. When dividing such adhesions it is always judicious to keep to the far side of the more important viscera to avoid injury. After the condition of the gall-bladder is noted, it may thus be used for traction to better expose the bile ducts. Surgery of the gall-bladder should be deferred until the surgery of the ducts is completed; with the surgeon on the left side of the patient, the common duct is palpated between the left index and middle fingers in the foramen of Winslow behind the common duct and the left thumb in front. Careful and gentle palpation is then made of the common bile duct both upward and downward, as well as of all the structures in the hepatico duodenal ligament. Knowing that the common duct normally lies anterior to the portal vein and to the right of the hepatic artery, its position is thus identified and the peritoneum over it divided. The duct by blunt dissection is now isolated. (It is most important that the common duct regardless of the level it is to be opened, be constantly kept in complete control between the fingers and thumb of the left hand.) To make sure that it is the common duct and not the portal vein, simple aspiration with a needle and syringe will confirm its identity. Two silk or catgut traction sutures are placed into the front wall of the common duct at the same level, far enough apart to permit an incision into the duct between them; with the duct and the stone, if present, held firmly fixed between the thumb and index finger the duct is incised longitudinally between the traction sutures with a sharp knife, while the tip of a suction stem keeps the field clean. The length of the incision will be determined by the size of the stone or stones. All are milked or aspirated from the duct or extracted with forceps. In the event a stone is impacted in the ampulla, or retroduodenal segment of the common duct, it may be dislodged by firm and sustained pressure. A narrow scoop

or sound passed down to the stone may aid in dislodging it, but their use is definitely limited. Should these measures fail, a more radical means must be employed (to be described later) to dislodge the stone. If the bile that escapes from the common bile duct is turbid or contains sediment, gentle irrigation of the duct through a small catheter may be employed. The ducts may then be probed, first upward toward the liver, then downward through the ampulla into the duodenum. This enables the surgeon to clear the lumen. If the bile is clear and devoid of sediment and all the stones have been removed, the incision may be closed without draining (Fig. 480, c). Most surgeons, however, employ a "T" tube and close the

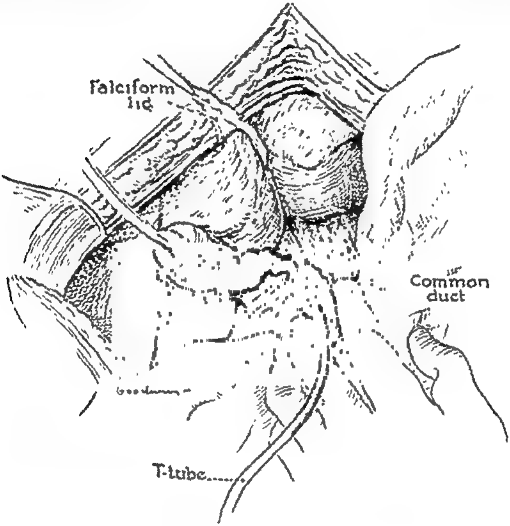

Fig. 481. — Shows a T-tube in the common duct and the manner of controlling the duct with fingers and thumb of the left hand.

duct around the tube with interrupted sutures (Fig. 481). The question now facing the surgeon is "What shall be done with the gall-bladder?" If the common duct is badly damaged by infection and ulceration and the cystic duct is patent, the gall-bladder should be drained, not removed. Should the healing and fibrosis cause stricture of the common duct, the gall-bladder will provide a most convenient means for a cholecystoduodenostomy. Some surgeons feel that the gall-bladder should be removed to prevent the recurrence of stones, that might again pass into the common bile duct. In the experience of the author, when stones recur in the common bile duct after drainage, they do so whether the gall-bladder is in or out. In most of these patients the biliary ducts are all greatly dilated and have a combined

Fig. 482.—Kocher's incision to mobilize the duodenum.

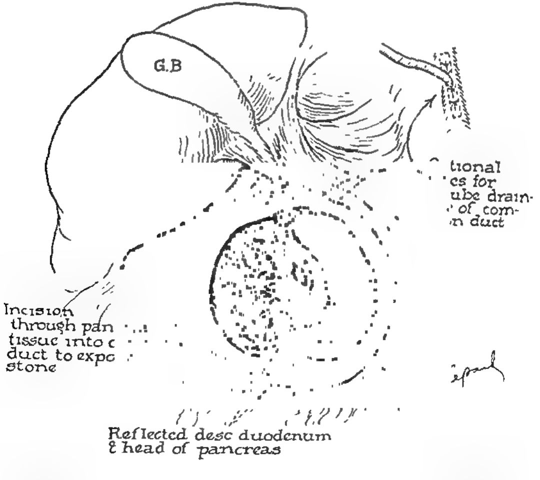

Fig. 483.—Shows mobilization of the duodenum in order to remove a stone from the retroduodenal or pancreatic portions of the common bile duct. This opening may be closed without drainage, or drainage with a T-tube, or it may be closed and a T-tube inserted in the extraduodenal portion of the duct if drainage is indicated

capacity much greater than the gall-bladder itself. Biliary stasis can therefore occur in the common duct as well as in the gall bladder.

Impacted common duct stones as already stated occur in either the retroduodenal segment of the duct or in the ampulla of Vater. In the event the stone is impacted in the retroduodenal segment of the duct, the duodenum is mobilized by incising the posterior peritoneum just above the second portion of the duodenum (Figs. 482 and 483). Rolling the duodenum forward and to the left exposes this segment of the duct which lies in a groove on the posterior surface of the pancreas or entirely surrounded by pancreatic tissue. The duct with the stone is held

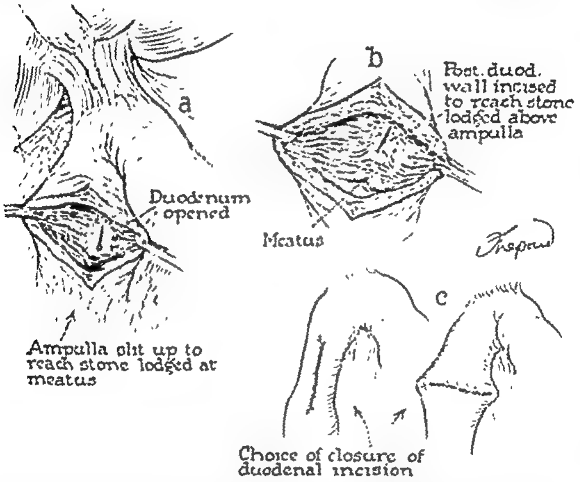

FIG. 481.—(*a*, *b*) Show transduodenal approach to ampullary and intramural portions of the common bile duct for removal of stone; (*c*) shows methods of closure of the duodenum. The tranverse suture is preferable if there is danger of greatly narrowing the duodenum.

snugly between the thumb and index finger of the left hand. An incision is now carried through the duct wall directly over the stone, which is then promptly expressed and the incision closed with silk or No. 00 chromic catgut. If drainage is necessary a "T" tube is introduced through the anterior wall of the common duct. To remove impacted stones from the ampulla, the transduodenal approach is utilized. This is done by first mobilizing the duodenum and then holding its second portion with the stone containing ampulla between the left thumb and index finger, a longitudinal incision 2 to 3 cm. long (Fig. 484, *a*) or a transverse one if desired is made in the anterior duodenal wall directly over the subjacent stone. This exposes the papilla which is made prominent by the bulging gall-stone. The orifice or meatus of the papilla may be dilated by introducing the closed end of a

Kelley artery forceps and then spreading the closed end enough to push the stone through. It may be necessary to cut the papilla (McBurney) to remove the stone (Fig. 484, *a*). In the event the stone is in the intramural portion of the duct (Fig. 484, *b*) an incision is made through the posterior duodenal wall over the stone through which the stone is expressed. A few interrupted stitches may be used for closure. The question may be asked "Should the common duct be now drained?" *It is the opinion of the author that whenever a stone has been removed transduodenally from the distal portion of the common bile duct requiring incisions in* the ampulla or the intramural portion of the duct a long T-tube of the Cattell type should be inserted. If the common bile duct has not already been opened at a higher level, such an opening should then be made for the insertion of the T-tube.

CHOLECYSTOGASTROSTOMY, CHOLECYSTODUODENOSTOMY AND CHOLEDOCHODUODENOSTOMY

Indications.—The purpose of either a cholecysto-gastrostomy or cholecystoduodenostomy is to provide a substitute channel for the passage of bile from the liver to the intestine by way of the *gall-bladder and cystic duct, when the common* bile duct has been rendered useless. This constitutes the so-called "by-pass" operation and can be used only if the gall-bladder is present and both cystic and hepatic ducts are patent. It is used largely as a palliative measure in inoperable malignancies of the common bile duct, and head of the pancreas. It may also be used *as a permanent measure in certain congenital* biliary duct atresias.

Choledochoduodenostomy, on the other hand, if it can be performed reëstablishes a more natural channel for the bile flow into the intestine, and should be used whenever possible. The indications for these three procedures mentioned above are:

1. Carcinoma of the head of the pancreas if inoperable and chronic recurrent *pancreatitis*.

2. Carcinoma of the common bile duct where resection cannot be achieved and where the cystic duct and common hepatic ducts are still patent.

3. Congenital cystic dilatation of the common bile duct. This condition is sometimes called "choledochus cyst" and occurs without any appreciable common duct obstruction. There may be *evidence of common duct stenosis, angulation* or a valve-like fold in the ampulla of Vater. When the "cyst" has once formed it may increase any angulation of the duct by virture of its weight, and increase the distension. In this condition a choledochoduodenostomy is the ideal operation.

4. Congenital atresia of the bile ducts. Only certain types of congenital atresias of the bile ducts may be relieved by cholecystoduodenostomy. This method is indicated only when the common duct is obliterated and the cystic and hepatic ducts are patent. In the event of atresia of the common and cystic ducts and a patent hepatic duct, reconstruction procedures are necessary.

Technique.—Cholecystoduodenostomy (Fig. 485). The same approach is employed as in cholecystectomy. The surgeon decides after exposure and exploration whether he will use the stomach or the duodenum for the anastomosis. Cholecystogastrostomy as a general rule is unsatisfactory, therefore, a cholecystoduodenostomy is preferable because the duodenum is so readily mobilized in spite of adhesions and when the anastomosis is completed, there is less traction on the anastomatic line. If the gall-bladder is enlarged and distended it is first collapsed by aspiration. Its fundus is then grasped with a curved forceps whose blades are

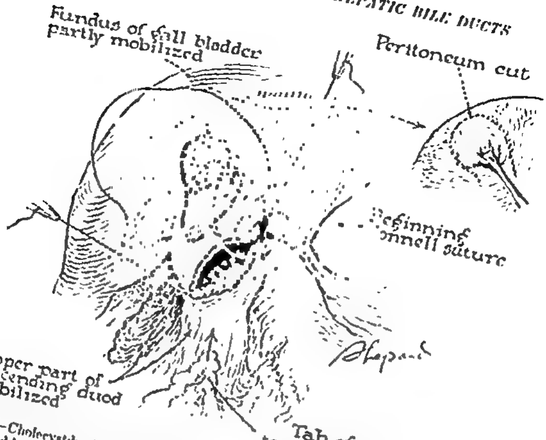

FIG. 485.—Cholecystduodenostomy (applicable also to cholecystagastrostomy). The fundus of the gall bladder and the duodenum have been mobilized (not always necessary). A posterior seromuscular Cushing right-angle suture has been placed, both organs have been opened, the Connell suture is being completed, after which the seromuscular suture will be completed anteriorly and the omentum tacked over the suture line.

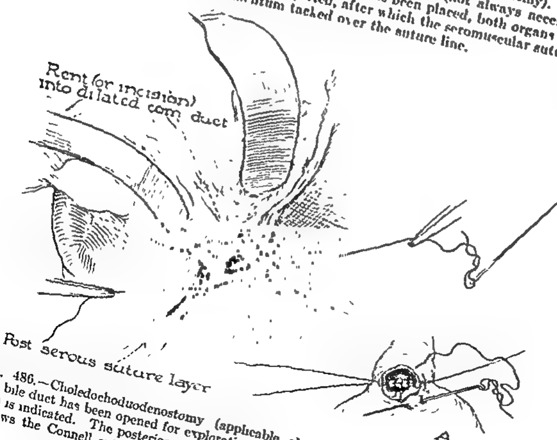

FIG. 486.—Choledochoduodenostomy (applicable also to choledochogastrostomy). The common bile duct has been opened for exploration and the site of incision in the duodenum (or stomach) is indicated. The posterior seromuscular Cushing right-angle suture has been placed. Inset shows the Connell suture being completed.

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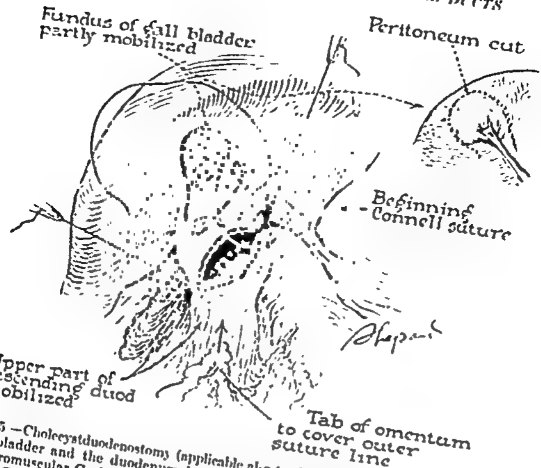

FIG 485.—Cholecystduodenostomy (applicable also to cholecystagastrostomy). The fundus of the gall bladder and the duodenum have been mobilized (not always necessary). A posterior seromuscular Cushing right-angle suture has been placed, both organs have been opened, the Connell suture is being completed, after which the seromuscular suture will be completed anteriorly and the omentum tacked over the suture line.

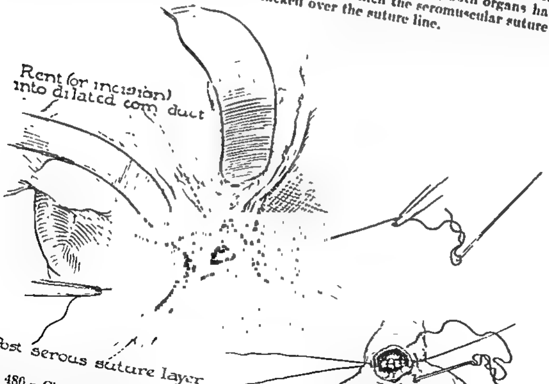

FIG. 486.—Choledochoduodenostomy (applicable also to choledochogastrostomy). The common bile duct has been opened for exploration and the site of incision in the duodenum (or stomach) is indicated. The posterior seromuscular Cushing right-angle suture has been placed. Inset shows the Connell suture being completed.

covered with rubber tubing. In the same manner the nearest portion of the duodenum is grasped and the two forceps are thus placed side by side. Laparotomy pads are then packed snugly around the clamps. A No. 00 chromic catgut or silk is used on a curved or straight needle to approximate the two serous surfaces with a running stitch for a length of about 3 cm. With a sharp knife an incision about two-thirds the length of the suture line is made into the gall-bladder wall and also into the duodenum close to and parallel with the suture line. A second chromic catgut suture is now used in running fashion through the cut edges of the two adjacent walls, following the opening around to the starting point where the ends are tied. This constitutes the inner row of suture of the anastomosis. The first chromic catgut suture is now taken up, the clamps removed from both viscera, and the suturing of the serosal walls is completed, constituting the outer row of

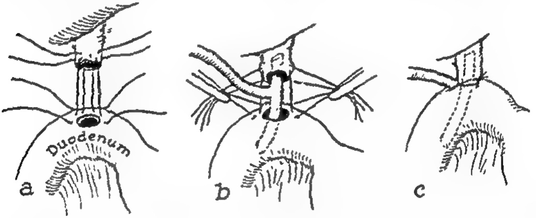

Fig. 487.—Shows a method of choledochoduodenostomy, described by Gatewood, which can be used if the duct has been severed or its lower portion is atresic. The anastomosis is made over a T-tube.

sutures. This completes the two row suturing of the anastomosis. Some surgeons prefer to do this anastomosis without the use of clamps, thereby avoiding any possible injury the clamps might inflict on the wall of either viscus. When the operation is done without clamps an interrupted anchor suture is placed through the serosal surfaces of both viscera and tied. A second similar suture is placed opposite the first about 3 cm. away to permit the anastomosis between the anchor sutures. The field is kept bloodless with the suction tip.

Choledochoduodenostomy.—This operation should be performed whenever possible instead of a cholecystoduodenostomy (Figs. 486 and 487). When the common bile duct is greatly dilated, as in congenital cystic dilatation, it provides ample surface for an easy side to side anastomosis with the duodenum. In the event that the common duct is not large enough to permit side-to-side anastomosis an end-to-side may be employed instead (Figs. 486 and 487). This procedure may be consulted under "Reconstruction of the Bile Ducts," Chapter 60.

CHAPTER 60

GALL-BLADDER AND EXTRA HEPATIC BILE DUCTS

By H. A. Oberhelman

RECONSTRUCTION OF THE BILE DUCTS

Indications.—In recent years so much progress has been made in reconstruction of the common bile ducts that the so-called "by-pass" or palliative operations which attack the obstructive lesions only indirectly have been more or less replaced by methods whereby the obstructive lesions regardless of cause are made the object of direct attack. Ever since W. H. Mayo in 1905 performed the first successful hepaticoduodenostomy, surgeons everywhere have become deeply interested in the reconstruction of the bile ducts and have made very noteworthy contributions in this important field. Some of the methods devised have been abandoned, some have yielded a fair degree of success, and still others have received quite general acceptance. In the same breath, it can be stated that no method so far devised is a completely satisfactory substitute for a destroyed common bile duct. It still

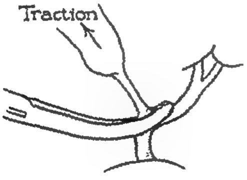

Fig. 488

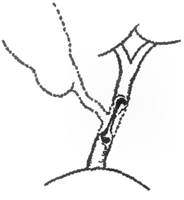

Fig. 489

Fig. 488.—Illustrates one of th[illegible] most comm[illegible] [illegible] bile duct—t[illegible]

Fig. [illegible] 488.

remains one of the most troublesome problems with which the surgeon has to deal. The causes of failure are not always mechanical, even though perfectly executed. Infection, such as cholangitis due to reflux of intestinal content and the depleted physical condition of the patient from jaundice or long standing loss of bile are, if not primary, certainly contributory causes for failure. Furthermore there are always some failures attributable to errors in judgment when attempts are made to remove a lesion that seems operable but proves to be inoperable. It must be freely admitted, however, that while these causes for failure can never be entirely eliminated, their incidence can and have already been reduced to a gratifying minimum.

The conditions for which reconstruction of the bile ducts are necessary will be given in the order of their frequency:

1. Benign strictures of the bile duct. The most common cause of benign stricture of the bile ducts is the result of some previous operative trauma, as distinguished from direct trauma due to violence. Operative trauma is inflicted either knowingly or unknowingly during operation on the gall-bladder and bile ducts. The injury may consist of complete or partial division of the common bile duct or a complete or partial ligation (Figs. 488 and 489) or in the event of uncontrollable hemorrhage may be clamped blindly. In a series of 23 patients with common duct strictures Cole and his associates[3] considered operative trauma the probable cause in 76 per cent. Cattell[4] found a similar cause in 80 per cent of his patients and a figure as high as 90 per cent is reported by Walters and Snell.[18] In a comprehensive study by Flickinger and Masson[7] of 188 patients with stricture of the common duct over a ten year period at the Mayo Clinic, 181 or 96 per cent had had cholecystectomy and 5 other patients had had some biliary tract surgery done, leaving only 2 patients without a previous biliary tract operation. In 73 per cent of the 188 patients operative trauma accounted for the stricture. The factors responsible for this important condition are as follows:

a. Lack of familiarity with the various anomalies of the bile ducts and blood vessels.

b. Needless opening of the common bile duct for purposes of exploration.

c. Removal of the gall-bladder from below upward instead of from above downward, (Allen).[1]

d. An incision which allows inadequate exposure of the operative field.

e. Hurry on the part of the surgeon (Cole). It can be readily appreciated that practically all of the causes just mentioned are avoidable, or at least could be reduced to a minimum if every surgeon maintains the greatest possible respect for the "simple gall-bladder operation."

2. Direct trauma to the bile ducts through external violence in civilian life is extremely rare. In a study by the author[8] of 313 patients with peace time bullet wounds of the abdomen, the common bile duct was injured but once. This wound was overlooked by the surgeon at operation but found by the pathologist at necropsy. Even the literature on wartime casualties likewise yields only rare instances of biliary tract injuries.

3. Inflammatory conditions causing benign strictures such as ulceration from gall-stones with subsequent healing and fibrosis, or a chronic pancreatitis with compression of the pancreatic segment of the common bile duct.

4. Benign tumors may occur but are very rare.

5. Congenital atresias while also rare are interesting because only a very small minority are operable. The great majority have no communication between the intra- and extra-hepatic ductal system. Since the jaundice in these atresias is always of the obstructive type and not of the hepaticocellular type, it is impossible to determine clinically whether the obstruction is in the intra- or extra-hepatic ductal systems. Therefore an exploratory laparotomy is imperative to determine the location of the atresia. Ladd and Gross found that in 45 exploratory laparotomies for biliary atresia in infants only 9 or 20 per cent presented a patent communication between the intra- and extra-hepatic ductal systems to allow intestinal anastamosis through hepaticoduodenostomy, choledochoduodenostomy or cholecystoduodenostomy. They found four different types of atresias (Fig. 490). From a study of the sketches it will be noted which types are operable and which types are

inoperable. In recent years the author encountered one each of the types B, C and D, with only one adaptable to surgery.

6. Carcinoma of the Bile Ducts. Carcinoma of the bile ducts, fortunately is relatively uncommon, but when it occurs it presents a surgical problem that carries with it great difficulties of execution, and equally as great operative risks. The infrequency of this lesion may be appreciated from a report made by Marshall[11] who found that out of 22,000 operations for lesions of the biliary tract at the Mayo Clinic only 41 were performed for carcinoma of the bile ducts, averaging slightly

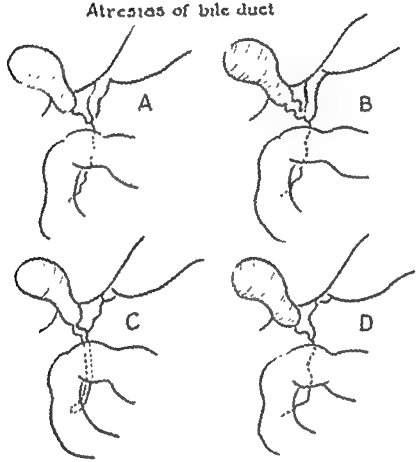

FIG. 490.—Congenital artesias of bile ducts.

less than 1 in 500 operations. The most common site of the carcinoma is at the junction of the cystic and the common bile ducts. A considerable number occur at the Ampulla of Vater. Gall-stones do not occur as commonly in carcinoma of the bile ducts as in carcinomas of the gall-bladder. The immediate spread of the carcinoma may be annular or longitudinal, and in the latter instance spreads by contiguity usually into the liver substance and renders complete removal impossible even though evidence of gross metastasis are not observed. In the event of an early carcinoma of the Ampulla of Vater, a Whipple operation has a definite place.

7. Carcinoma of the Head of the Pancreas. While this lesion is not a primary lesion of [illegible]
lesion of [illegible]
Ampulla of Vater or of the head of the pancreas may all be regarded as one and the same entity as far as surgical therapy is concerned for all are treated by the same surgical procedure.

TECHNIQUE OF RECONSTRUCTION OF THE BILE DUCTS

In considering the surgical technique for the reconstruction of the bile ducts one is overwhelmed by the many different methods that have been devised for this purpose since W. H. Mayo reported the first successful hepaticoduodenostomy in 1905. This was to be expected since obstructive lesions of the bile ducts have been and still are a universal problem. Many noted surgeons the world over have sought a solution for this problem, and as a result a variety of solutions have been offered. Furthermore, the variety of these obstructive lesions and the various levels at which they occur, create conditions that obviously cannot be treated by one and the same technical procedure. This also has accounted in part for the many different methods of repair offered. In many instances the solution might be considerably more simple if it had not been for the fact that most of these patients with biliary duct strictures have had a previous cholecystectomy, and are thereby deprived of the only structure nature intended as a substitute for the common bile duct. However, it is quite obvious that any reconstructive surgery of the bile ducts represents a type of surgery that is difficult and beset with many problems and meets with only a fair degree of success in the hands of the best surgeons. It might be timely to advise surgeons whose experience with this particular field of surgery has been limited that such patients be referred to those whose experience has been more extensive. These patients are all serious surgical risks with a high mortality and since there is no alternative method, these patients all fall into the so-called group of "must surgery." Patients who are deeply jaundiced may require a two-stage operation, the first of necessity to relieve the biliary obstruction so that the second and more prolonged reconstructive operation may be more safely done. For the same reason those patients with a long standing external biliary fistula with loss of bile must have a disturbed metabolism restored to a normal balanced metabolism.

It is not the author's purpose to present in detail all of the various methods of reconstructive surgery ever devised. The success of any method according to Allen[1] in restoring continuity between the biliary and gastrointestinal systems depends upon a wide, free permanent communication. The following methods for the most part meet such requirements.

1. **End-to-End Anastomosis.**—This is the simplest method of reconstructing the common bile duct. Unfortunately it is applicable to only a small minority of cases. When the duct is freshly divided either through surgical accident or penetrating violence, end-to-end anastomosis is the ideal method. It is also useful in the event of any local lesion such as a small tumor or a stricture when only a short segment of the duct is involved. The anastomosis may be made with the ends of the duct directly approximated or it may be made over a rubber tube whose distal end extends through the ampulla into the duodenum (Fig. 491).

Sullivan[15] was the first to employ a tube over which the anastomosis could be made. C. H. Mayo advocated this method and provided a tube known as the Mayo-Sullivan tube (Fig. 492). McArthur[12] employed a tube provided with a cuff (Fig. 491). In addition, C. H. Mayo substituted a T-tube for the straight tube bringing the long arm of the T-tube out at the level of the anastomosis. Instead of bringing the T-tube out at the level of the anastomosis Lahey[10] advised that it be brought out just above the level of the anastomosis through a new opening in the duct (Fig. 492b). The healing of the line of anastomosis will not be interfered

with; Cattell[4] shares the same view but brings it out from the common duct below the level of the anastomatic line (Fig. 492c.)

2. **Choledochoduodenostomy and Hepaticoduodenostomy.**—Because end to end anastomosis is applicable only to a limited number of duct lesions, the method of establishing a direct anastomosis between what remains of the common hepatic or common bile ducts and the intestine is the method of choice. The method devised by W. H. Mayo and which has been so extensively used by Walters,[17]

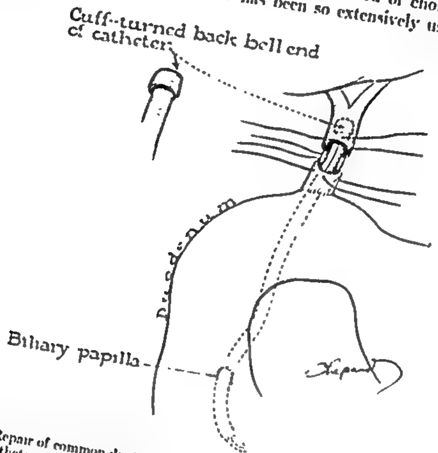

Fig. 491.— Repair of common duct (McArthur technic). A cuff is turned at the "bell" end of a soft rubber catheter, this end is inserted into the proximal portion of the duct, and the "eye" end is passed into the distal segement and out through the sphincter of Oddi into the lumen of the duodenum. The cuff aids in keeping the catheter longer in place. The duct is then anastomosed over the catheter and the suture line reinforced with omental fat.

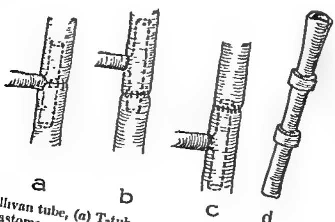

Fig. 492.—Mayo-Sullivan tube, (a) T-tube emerges at the level of the anastomosis; (b) tube comes out above the anastomosis; (c) tube emerging below the level of the anastomosis, (d) Sullivan tube

provides for a valve-like structure that *prevents an ascending* cholangitis. The technique is as follows: The usual incision for biliary tract surgery is made. The extensive adhesions from a previous cholecystectomy and attempts at surgical repair are carefully divided. Since the patient is frequently jaundiced or in a depleted metabolic state, bleeding is an annoying factor. Lines of cleavage should be carefully followed, using the undersurface of the liver as a guide. Having by blunt and sharp dissection exposed the sub-hepatic space the stomach, duodenum and the stump of the cystic duct are identified. With traction on the stump of the cystic duct and with the left index and ring fingers in the foramen of Winslow, *if not*

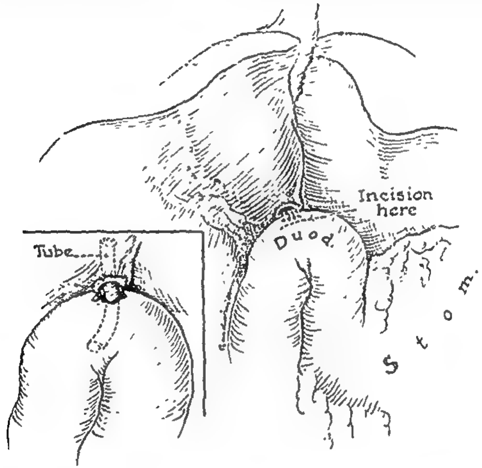

Fig. 493.—Anatomosis of severed end of hepatic or common duct to the duodenum. Inset shows a straight Mayo-Sullivan tube in place.

obliterated by adhesions, dissection is then carried upward and downward in the hepaticoduodenal ligament in search for the common or common hepatic bile ducts. If a fistulous tract is present, the identification of the ducts is greatly facilitated. Great care must be exercised to avoid injury to major blood vessels and anomalous ducts, although it is quite probable that any anomalies, if present, were destroyed at the first operation. The dissection may reveal any one of several conditions

1. There may simply be an incomplete stricture of the bile ducts.

2. The upper end of the common or common hepatic duct may consist of a bulbous enlargement and have no communication or connection with the lower end of the common duct.

3. The stricture may be in the distal end of the common duct, and if so the common duct may easily be mistaken for the duodenum because of its immense size.

If any difficulty is encountered in distinguishing between the common bile duct and the portal vein an aspirating syringe with a fine needle will determine which is which. Search should always be made for the proximal end first, the end to be used for the anastomosis. The distal end is usually represented by a cord which is valueless and probably should not even be searched for. Having isolated the proximal end of the common duct or common hepatic as the case may be, the duodenum is brought up to the open end of the duct for an end-to-side anastomosis

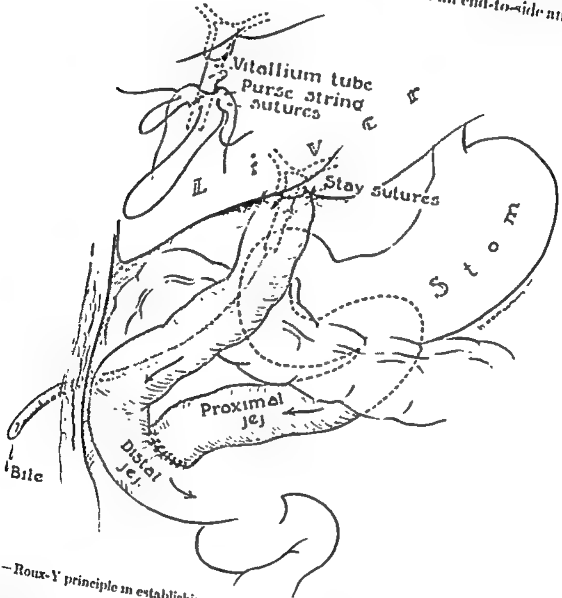

FIG. 491.—Roux-Y principle in establishing continuity between the bile ducts and the jejunum.

and held with stay sutures. A semilunar incision 2 to 3 cms. long with the concave side anterior is made through the duodenal wall. The curved flap thus made in the duodenum is then sutured to the posterior edge and sides of the open duct with interrupted silk making a muco-mucous approximation. Before completing the anastomosis a Mayo-Sullivan tube is then introduced—the bell-end directed upward into the duct, the lower end downward into the duodenum for 2 to 3 cm. and fixed with slowly absorbable suture material The anastomosis is now completed anteriorly. Since the opening in the duodenum is larger than the open duct, the unused segment of the duodenal opening is closed by suturing it either to the

under surface of the liver through Glissons capsule if the anastomosis is at the liver hilus, or it may be closed with interrupted sutures. This usually allows for an opening less likely to end in stricture. The free margins of the omentum are sutured around the anastomosis to prevent leakage and hasten repair. When the sutures holding the Mayo-Sullivan tube are absorbed after several weeks the tube passes readily into the intestine and is eliminated.

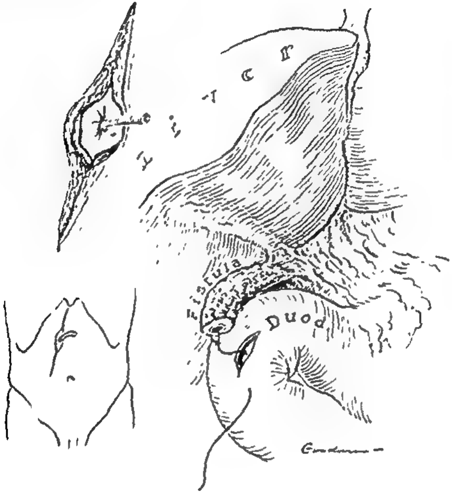

FIG. 495.—Illustrates transplantation of fistulous tract into duodenum.

Jacobson[9] found that when the duodenum could not be mobilized adequately because of adhesions, a loop of jejunum could be employed instead. In order to more fully overcome strictures and reflux of intestinal content into the bile duct, Whipple[20], Allen[1] and Cole and associates[5] have made use of the Roux-Y principle in establishing continuity between the bile ducts and intestine (Fig 494). Pearse[14] advised the use of a vitallium tube to be left in permanently to avoid stricture at the level of the anastomosis. He favored this type of tube because of less encrustation in the duct at the site of the anastomosis. Cole and his associates[5] also favor the vitallium tube and report good results using it with Roux-Y principle. On the other hand Allen[1] definitely opposes the use of any type of permanent tube because of eventual occlusion from deposition of bile salts. Instead he recommends the use of a temporary tube in the Roux-Y principle. Wilson[22] advocated a serous lined tube from the stomach and reported a successful case. In much the same way Dragstedt and his associates[6] favored the use of the duodenum.

3. **Transplantation of an External Biliary Fistulous Tract into the Duodenum.**—Transplantation of an external biliary fistulous tract into the duodenum has been found successful in certain selected cases, and represents a relatively simple technical procedure (Figs. 495 and 496). It was apparently first attempted in the human by Sutton[11] but unsuccessfully, although V. Stubenrauch (1906) had successfully employed the principle in experimental animals previously. Williams,[21] however, successfully implanted such a biliary fistula into the duodenum of a young boy four years old, a fistula created shortly after birth for atresia of the common bile duct. According to Walters if the biliary fistula discharges all of the bile excreted and resists all attempts at closure, the transplantation of such a fistula into the stomach or duodenum should be successful. The technical procedure consists of first passing a catheter into the tract as far as it will go. The tract is then dissected out leaving a generous wall of surrounding tissue attached. When this is adequately mobilized, a stab hole is made into the duodenum or stomach, preferably the duodenum. A

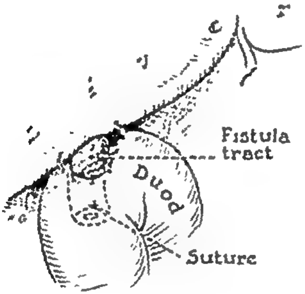

Fig. 496.—Fistulous tract inserted into the duodenum and held in place with interrupted sutures.

silk suture is now passed through the end of the fistulous tract, tied, then passed into the duodenal opening and brought out again about 2 to 3 cm. lower down. This suture is used to draw the fistulous tract into the duodenum where the edges of the duodenal opening are sutured snugly to the outside of the fistulous tract.

4. **Carcinoma of the Bile Ducts.**—The surgical procedures already described for the treatment of benign strictures are equally applicable to malignant strictures, provided *the involvement is local and in the supraduodenal portion of the* common duct. This is rarely the case because no early clinical signs of carcinoma of the bile ducts appear to lead to early surgery. Biliary obstruction, however, eventually develops and when signs of jaundice appear the condition has invariably advanced to the inoperable stage. In the experience of the author most carcinomas of the bile ducts have already reached this stage and that only palliative procedures are indicated such as: cholecystoduodenostomy if the cystic and hepatic ducts are patent; or inserting a tube into the hepatic duct if an uninvolved segment can be found. When the carcinoma involves the ampulla or periampullary region it is difficult to

determine clinically or at operation the starting point; whether in the ampulla, the terminal part of the common bile duct, or the pancreatic duct. Regardless of their starting point, all are identical so far as any surgical therapy is concerned. Therefore in recent years a strong tendency has developed toward a more radical resection, and to now regard inoperable conditions as operable. Whether these more radical procedures are justifiable in the final analysis may not be generally too convincing. However, there can be no question about their justification if they are performed in the operable stages of the disease. They carry a grave risk but offer the only chance for a cure. Whipple and his associates[10] have devised a radical resection requiring two stages for ampullar and peri-ampullar malignant lesions. In the first stage the common duct is ligated and an anti-colic cholecystojejunostomy according to the Roux-Y principle is completed. The second stage consists of a posterior gastrojejunostomy and a resection of the duodenum and head of the pancreas. Brunschwig[2] likewise has devised a two-stage resection of the Ampulla. The first stage consists of a posterior gastroenterostomy, an anastomosis of the gall-bladder or stump of the common duct to a loop of jejunum behind the transverse colon and an enteroenterostomy between the efferent and afferent loops of the jejunum. In the second stage, the common duct is ligated (if not used for a choledochojejunostomy in the first stage) and the duodenum with the head of the pancreas is resected. More recently Brunschwig[2] has succeeded in accomplishing a pancreaticoduodenostomy in one stage.

General Results.—In summarizing, the results generally of cholecystostomy and cholecystectomy have been good. Cholecystectomy for calculous cholecystitis has been far more uniformly satisfactory than cholecystectomy for non-calculous cholecystitis. If symptoms persist after cholecystostomy or cholecystectomy it is embarrassing to the surgeon and distressing to the patient. These persistent symptoms may be attributed to any one or more of several factors: (1) A small stone or several may have been overlooked at the time of surgery. This can easily occur if the gall-bladder and cystic duct are filled with multiple small stones; one or more can easily escape from the cystic duct into the common duct during removal of the gall-bladder without the knowledge of the surgeon. To have overlooked a stone is the surgeon's first reaction when symptoms persist. (2) Much has been written about the spasm of the sphincter of Oddi causing a resistance greater than what the secretory pressure of bile can overcome. The pain presumably results from over distension of the bile ducts. (3) A persistent chronic pancreatitis which may be painful *per se* or may tend to compress the common bile duct but not occlude it. (4) Injury to the common bile duct either through incomplete or complete ligation or excision or both. Under such conditions jaundice will be progressive and reoperation will be necessary. (5) In case of a mistaken diagnosis where the symptoms were functional rather than organic and cholecystectomy afforded no relief. Death after cholecystectomy of unexplained etiology is known as the so-called "liver deaths." Henschen[1] describes a group of patients whom he chooses to designate as "liver weaklings" because they seemingly do not possess adequate liver function to meet the additional strain demanded in major biliary tract surgery. Allen is of the opinion that some of the so-called "liver deaths" are no doubt the result of ligation of the right hepatic artery instead of the cystic artery. However, with our increased knowledge of liver functions during recent years, of a much more intelligent preoperative program devised to adjust and correct deranged liver physiology, and of a more comprehensive knowl-

edge of the anomalies of biliary ducts and blood vessels, such deaths now rarely ever occur.

Surgery of the bile ducts so far as stones are concerned has been uniformly good. For tumors, both benign and malignant, radical resections have yielded some encouraging results if seen in the earlier stages of the disease. When the ampullary region is involved, only the more radical procedure—such as a pancreatico-duodenectomy while grave, has yielded favorable results.

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CHAPTER 61

SURGERY OF THE PANCREAS

By John L. Keeley

GENERAL CONSIDERATIONS

The pancreas, lying deep in the recesses of the upper abdomen has been the subject of much investigation during the past ninety years. Its complex structure and functions have been discovered by brilliant research, so that its roles in carbohydrate and fat metabolism and in intestinal digestion are now well established. In a discussion confined to the surgical diseases of the pancreas it is impossible to elaborate upon these great discoveries, but it must be acknowledged that successful surgery of the pancreas has utilized them particularly in relation to carbohydrate metabolism and the functions of the external secretion of the gland.

EMBRYOLOGY

The pancreas is formed from two buds at the level of the future duodenum. The dorsal (proximal) bud is the larger of the two and arises directly from the upper part of the future duodenum. The ventral (distal) pancreatic bud arises

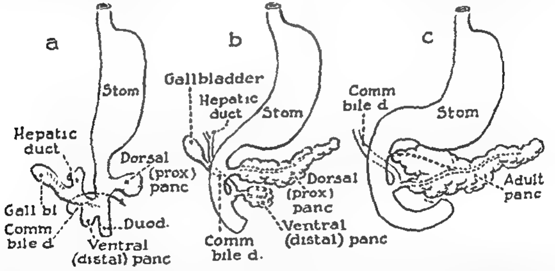

Fig. 497.—Stages in the development of the pancreas. The rotation of the ventral pancreatic bud (a) brings it into position to fuse with the dorsal pancreatic bud in (b) to form the adult pancreas as in (c). The embryological basis for the relationship of the common bile and main pancreatic ducts is shown.

from the future common bile duct, which is a part of the hepatic bud. This smaller pancreatic bud rotates to the right and posteriorly behind the duodenum, eventually fusing with the dorsal pancreatic bud to form the fully developed gland (Fig. 497). The common bile duct and the main pancreatic duct (Wirsung) fuse. The distal end of the duct of the smaller ventral pancreatic bud joins the duct of the dorsal

pancreatic bud at a variable point in the body of the pancreas, resulting in a Y configuration, the upper arm of the Y representing the accessory duct of Santorini and the lower arm representing the main pancreatic duct of Wirsung (Fig. 497). Variations in development may lead to anomalies in the formation of ducts.

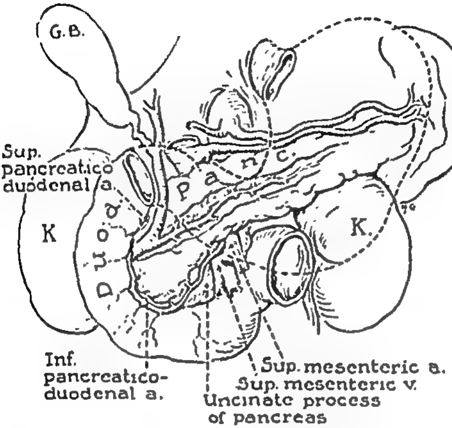

FIG. 498.—Gross anatomy about the head of the pancreas and the duodenum. The relationship of the uncinate process to the superior mesenteric vessels is shown, as well as the blood supply to the head of the pancreas and adjacent duodenum.

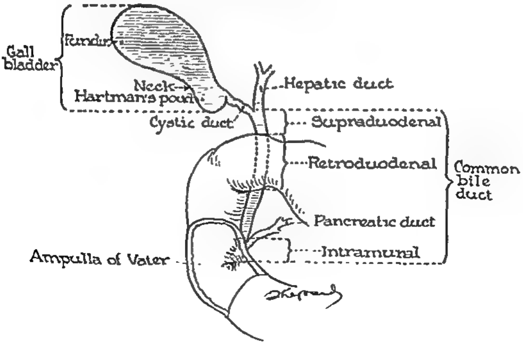

FIG. 499.—Diagramatic illustration showing the five divisions of the common bile duct.

As the duodenum encloses the head of the pancreas, the superior mesenteric artery about which the gut rotates comes to rest behind the body of the pancreas at its junction with the pancreatic neck (see Gross Anatomy; also Fig. 498), passing forward and downward to lie in front of the uncinate process of the head of the pancreas. About 3 cm. of the common bile duct thereafter lies in a groove between the duodenum and the head of the pancreas (one-third of cases), or in a tunnel in the head of the pancreas (two-thirds of cases), and is exposed to pressure by tumefaction in this part of the gland (Fig. 499). The origin of the main pancreatic duct in the hepatic bud makes possible the anatomic arrangement by which spasm of the sphincter of Oddi or obstruction in the ampulla of Vater permit reflux of bile and pancreatic secretion from one duct to the other, depending on intraductal pressures (*Fig. 500*).

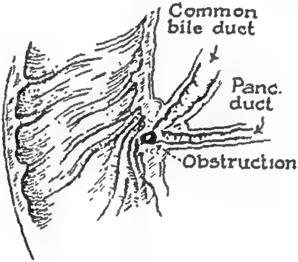

FIG. 500 —Obstruction in the ampulla of Vater permitting reflux of pancreatic or biliary secretions

The rotation of the gut with the dorsal and ventral mesogastrium forms the lesser sac of the peritoneal cavity. Part of the posterior wall of the lesser sac is the peritoneum covering the pancreas. Exudate from this peritoneum arising in response to inflammatory processes in the pancreas may accumulate in the lesser sac, if the foramen of Winslow has been sealed off by an inflammatory process.

GROSS ANATOMY

The pancreas is an elongated J-shaped digestive and endocrine gland lying transversely in a retroperitoneal position in the upper abdomen at the level of the first and second lumbar vertebrae. It is divided into head and neck, which lie *essentially in the midline and to the right in front of the aorta,* and body and tail, which lie to the left of the midline. The tail is in close relationship to the hilum of the spleen, the greater curvature of the stomach and the hilum of the left kidney. The normal pancreas weighs approximately 80 gm., is 12 to 15 cm. long *in situ* and has a lobulated appearance which resembles that of the larger salivary glands.

Since pathologic processes in the pancreas are characterized by effects on neighboring structures, the anatomy which makes this possible deserves emphasis. Thus, accumulations of fluid in the lesser sac displace the stomach forward, as in acute pancreatitis or in pseudocyst of the pancreas and cysts or large tumors of the

pancreas itself (Figs. 501, 502, 503, 504). It may be displaced upward or downward, depending upon whether the mass presses toward the gastrohepatic omentum or toward the greater omentum at the greater curvature of the stomach.

Due to the intimate relationship between the head of the pancreas and the duodenum, tumors in the head of the pancreas or enlargement of the lymph glands in this area may distort or enlarge the duodenal curve, as demonstrated by *x*-ray

FIG. 501.—Lateral view showing stomach and duodenal loop outlined by barium. Accumulation of fluid in lesser sac displaces stomach superiorly and anteriorly.

FIG. 502.—Upper abdominal mass in a case of pseudocyst of pancreas.

studies with barium (24). The same pathologic processes may lead to *external* compression of the common bile duct, resulting in obstructive jaundice with dilation of the bile ducts and gall bladder (Courvosier's law). As pointed out under Embryology, the common bile duct and the main pancreatic duct may have a common opening into the duodenum. There seems to be fairly general agreement that in approximately two-thirds of instances, the lumina of the common bile duct and the main pancreatic duct can be converted into one continuous passage by ampullary obstruction (Fig. 500).

The blood supply of the pancreas is derived principally from the splenic artery through its pancreatic branches and from the superior mesenteric and hepatic arteries by the inferior and superior pancreato-duodenal arteries which form a loop running around below and to the right of its head (Fig. 498).

Fig 503.—Opening of fistulous tract following marsupialization of pseudocyst of the pancreas.

The lymphatic drainage of the pancreas is toward the glands at the root of the superior mesenteric artery above and below the neck of the pancreas. These lymphatics drain ultimately into the celiac glands.

The nerves of the pancreas are branches of the celiac plexus which accompany the arteries to the gland. Just behind the gland lies the main part of the celiac plexus. Due to involvement of this plexus, many lesions of the pancreas are associated with pain radiating into the back. In fact, back pain in the recumbent position is highly suggestive of tumor of the body or tail of the pancreas (24). A fair correlation between back pain and encroachment on these nerve fibers has been found[14].

CONGENITAL ANOMALIES

There are only two congenital anomalies of the pancreas which are of importance: heterotopia, or aberrant pancreatic tissue, and annular pancreas. The former is not uncommon. More than 100 additional cases reported since 1946 (1) bring the current total to approximately 600. These aberrant pancreatic masses, 70 per cent of which are located in the stomach, duodenum or upper jejunum, are usually single. They are yellow or white opaque, lobulated masses, 1 cm. to 4 cm. in their greatest dimension, and round or irregular in shape. In relation to the layers of the intestine, about 60 per cent are located in the submucosa, about 25 per cent between the muscular layers, and only 4 per cent are subserosal. Their importance lies in the fact that their true nature may not be appreciated at laparotomy and an unnecessarily extensive operation may be performed because of a mistaken diagnosis of malignancy. Aberrant pancreatic tissue may cause pyloric or intestinal obstruction by simple lumenal encroachment or, in the small bowel, by intussusception. This tissue is subject to inflammation, ulceration, cystic changes and hemor-

rhage like the normally located pancreas. Six cases of hyperinsulinism in aberrant pancreatic tissue have been reported (1). This possibility should be kept in mind when Whipple's triad* is present and no tumor has been found in the pancreas.

Annular pancreas is a rare condi[illegible] cases to be reported up to the present time (24).

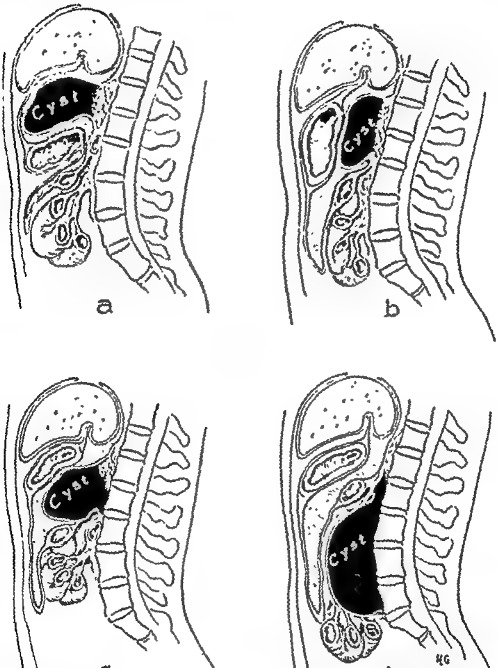

FIG 504.—Diagram showing pseudocysts of the pancreas presenting; (*a*) above the stomach; (*b*) posterior to the stomach; (*c*) below the stomach; (*d*) in the base of the mesentery.

due to the fusion of the proximal portions of the ventral and dorsal pancreatic buds (Fig. 497) in front of the duodenum and the normal fusion of the more distal portions of these buds behind the duodenum, thus forming a complete circle or ring of pancreatic tissue about the second portion of the duodenum. In some instances,

*Attacks of insulin shock coming on during fasting or in an overfatigued state, blood sugar levels of 50 mg. per cent or less, and relief by ingestion or administration of dextrose.

the ring may be incomplete, due to a cleft anteriorly. The complete ring, or annular pancreas, is associated with a high degree of duodenal obstruction. Rarely is there jaundice, due to the compression of the common bile duct. When annular pancreas causes duodenal obstruction in the newly born, it is usually diagnosed as duodenal atresia and is accompanied by persistent vomiting. There are no reported cases in the group between infancy and the late teens. In adults, annular pancreas has been erroneously diagnosed as chronic ulcer, diverticulum, congenital band, primary neoplasm or duodenal ileus. The correct preoperative diagnosis was first reported by Laymen in 1942. Isoperistaltic gastrojejunostomy is the preferred treatment, according to Gross and Chisholm ([20]), but Goldyne and Carlson ([19]) point out that five adult patients have had division of the ring without mortality and with cures in 4 of the 5 cases. The fifth was not completely relieved. A total of 71 cases have been treated surgically ([29]).

PHYSIOLOGY

The physiology of the pancreas concerns both its internal and external secretions. The principal internal secretion is, of course, insulin, which arises from the beta cells of the islets of Langerhans. It is responsible for the metabolism of carbohydrates. A deficiency in this secretion is the fundamental disturbance in diabetes, while over-production or excess of insulin (hyperinsulinism) causes a hypoglycemic state and may be due to diffuse hyperplasia of the pancreas or to benign or malignant lesions of the islets of Langerhans. Dragstedt ([12]) has recently demonstrated another internal secretion of the pancreas which, because of its importance in the utilization of fat, has been named lipocaic, a term derived from two Greek words meaning "I burn" and "fat." This substance is found in a fat-free alcoholic extract of pancreas and possibly arises from the alpha cells of the islets of Langerhans. It has been shown that it prevents the fatty infiltration and degeneration of the liver which occurs in depancreatized dogs. These changes do not occur when the external pancreatic secretion is prevented from entering the gastro-intestinal tract (as by the construction of a pancreatic fistula) and the pancreas remains normal. However, when the pancreatic ducts are ligated and the pancreas undergoes atrophy (often affecting both the parenchyma and the islets), fatty changes of moderate degree occur in the liver of a minority of the animals, due presumably to a decrease in the internal secretion rather than to exclusion of the external secretion from the gastro-intestinal tract.

The external secretion of the pancreas is a clear, watery, alkaline fluid with a specific gravity of about 1.015 and a pH which varies from 8.7 to 8.9. The amount produced daily ranges from 500 to 1500 ml., depending upon various stimuli. It contains three important enzymes: trypsin, amylase and lipase, which are proteolytic, amylolytic and lipolytic, respectively.

Exclusion or absence of the external pancreatic secretion from the intestine results in the production of bulky, greasy stools, because 30 to 70 per cent of the ingested nitrogenous material and 40 to 60 per cent of the ingested fat appear in the feces totally undigested. In the newly born, meconium ileus has been shown by Farber ([16]) to be due to lack of external secretion of the pancreas in cases of fibrocystic disease of that organ. The importance of an unimpeded outlet for the external secretion of the pancreas, as shown by Dragstedt's work, has a practical application in duodenopancreatectomy. In the early cases, no effort was made to anastomose

the pancreatic duct to the gastrointestinal tract, and some patients developed severe nutritional disturbances postoperatively. Since it has become apparent that there is no way of knowing which patients will suffer nutritional disturbances from this arrangement ([45]), renewed interest in methods of implanting the pancreatic duct into the jejunum has arisen. Pearse ([35]) states that the greatest benefit from re-establishing continuity is the decreased liability of pancreatic fistulæ.

ACUTE PANCREATITIS

Both etiology and classification of this disease have been the source of considerable confusion. At the risk of oversimplification, the following resume, based on studies summarized by Cole ([7]), is offered. Inflammation of the pancreas starts with rupture of tiny intraglandular ducts, permitting the pancreatic ferments to come in contact with the parenchyma. Rupture of the ducts may be brought about by back pressure in the ductal system caused by 1) spasm of the sphincter of Oddi, 2) stone at the ampulla of Vater, 3) metaplasia of the duct epithelium ([37]), 4) reflux of bile under increased pressure, 5) increase in pancreatic secretion due to ingestion of heavy meals or alcohol, 6) localized edema near the head of the pancreas due to infection ascending the ducts, or a combination of two or more of these factors. The rôle of trauma from penetrating and non-penetrating wounds of the abdomen is at once obvious in producing so-called traumatic pancreatitis.

The severity and duration of the initial damage will determine to a great extent the severity of the pancreatitis. The lesser degrees of pancreatitis are called *acute interstitial* or *acute edematous* pancreatitis. There is a second group which McWhorter ([29]) has divided into *hemorrhagic, necrotic* and *suppurative*. The latter are thought to represent more severe degrees of the same process and, indeed, their clinical manifestations parallel the pathologic changes in severity and duration. The erosion of capillaries ([22]) and the added factor of infection account for the hemorrhagic and suppurative types. The amount of swelling which occurs so promptly in the gland causes areas of ischemia, and thus the necrotic type develops.

Whatever the most important factor in the pathogenesis of acute pancreatitis, the fact remains that the various stages or degrees thereof are associated with gall bladder disease in as many as 60 per cent of the cases ([13]). Jones ([25]) found gall stones in 49 per cent of a series of 43 patients with acute pancreatitis. Further proof of this association is supplied by the rarity of attacks of acute pancreatitis after cholecystectomy, the few exceptions being those instances where there has been a continuation or residue of an infection in the pancreas present at the time of operation.

Concerning [illegible] quent activatio [illegible] pressure in the pancreatic ductal system is greater than the secretory pressure which develops in the bilary tract ([12,37,53]). (Whether this difference is present in patients with gall bladder disease or with spasm of the sphincter of Oddi has not been determined.) It is significant that clinically, acute pancreatitis usually develops two to three hours after a meal and that the injection of a small amount of bile into the pancreatic ducts will be followed by acute pancreatitis only when done about three hours after a heavy meal.

Clinical Manifestations.—The outstanding findings are epigastric pain, nausea and vomiting. The severity of these symptoms varies with the extent of the patho-

logic changes. The attacks (usually recurrent; 37 per cent of McWhorter's series) usually begin two to three hours after a heavy meal, and although the symptoms develop rapidly, their onset is rarely as sudden as those of perforated peptic ulcer, with which the lesion is most often confused. Shock may be present within a few hours after onset. Vomiting is liable to be persistent; pain radiates to the left and through to the back. The tenderness is confined early to the area of the pancreas, but later, when peritonitis develops, it becomes more widespread and is accompanied by muscle spasm. Except for the absence of generalized board-like rigidity of the abdominal wall, the clinical picture so strongly suggests perforated peptic ulcer that Vaughn ([46]) has used the following figure of speech: "Acute pancreatitis is a ruptured ulcer in a fat patient, without a gas bubble (pneumoperitoneum)." Discoloration of the abdominal wall means extraperitoneal spread of blood and pancreatic secretion, but it is not common ([10]). Fever and leukocytosis develop, if the process persists, and the blood amylase may reach as high as 1000 units, from a normal of 90 to 150 units.* In the diagnosis of late cases it is well to remember that elevated amylase values in peritoneal fluid persist for two to four days after the serum values return to normal. The rise may be absent in the more fulminating forms. A decrease in total blood volume has been found in the more severe cases and restoration of normal blood volume by either serum albumen or whole blood is often necessary to correct shock. Serum calcium levels are diminished in acute pancreatitis between the second and fifteenth days, being lowest about the sixth day. Signs of tetany such as Chvostek's and Trousseau's signs may be present. Serum calcium levels below 7 mg. per 100 ml. seem to occur in fatal cases only. Glycosuria occurs in 10 per cent of cases. Diabetes may occur during acute pancreatitis ([41]) or develop later (in from 3 to 10 per cent of cases). Studies of carbohydrate metabolism should be done during the course of the disease and also as part of the follow-up study.

Treatment.--The trend at present is toward conservative management, if the diagnosis can be made. Surgery is therefore reserved for the treatment of the associated gall bladder disease (see Chapter on Surgery of Gall Bladder and Extra Hepatic Bile Ducts) and for the management of hematoma, pancreatic abscess or pseudocyst. If the first two are drained or if the latter is marsupialized (See Figures 501, 502 and 504) pancreatic fistulæ may form (see Fig. 503). Spontaneous healing may occur, but many of these must be dissected out and transplanted in a portion of the gastrointestinal tract ([27,28]).

More recently effort has been directed towards internal drainage of pancreatic cysts by means of a pancreaticocystojejunostomy using a lateral anastomosis or Roux-Y anastomosis.[33] A selection of this mode of management is based on the location of the cyst, its unresectability, the inadvisability of resecting the cyst-bearing portion of the pancreas and the nature of the cyst wall. The advantages of the Roux-Y anastomosis are those associated with freedom from regurgitation of intestinal contents into the cyst with possible infection of the cyst and to some extent the pancreatic tissue immediately surrounding it.

TRAUMATIC PANCREATITIS

Due to the deep location of the pancreas and its intimate anatomical relationship with other organs, isolated injury of the pancreas is extremely rare. Schmieden

* Somogyi's method

and Sebening ([4]) in 1928 were able to find but 20 cases of isolated injury of the pancreas in the literature. They further reported that in their study of 2,137 cases of acute pancreatitis, only 62 were due to trauma. Only 1 in Truesdale's 51 cases ([4]) and one in the series of Shallow, Eger and Wagner ([2]) were of traumatic origin. Naffziger and McCorkle ([3]) reported 5 cases of traumatic pancreatitis. All were associated with injuries to adjacent organs. Usually the associated injuries involved the spleen, liver, stomach, kidney, gall bladder, common bile duct, aorta, vena cava, diaphragm or lung. Injury to the pancreas may be due to penetrating trauma as from bullets, knives and other sharp objects. Non-penetrating injury may be due to falls, kicks, blows or crushes. Damage to the pancreas may result from operative procedures such as biopsy, partial pancreatectomy, operations on the stomach, spleen, left kidney or common bile duct.

The occurrence of upper abdominal trauma should arouse suspicion of injury to the pancreas, but because of associated injuries, there is no diagnostic clinical picture of pancreatic injuries. The diagnosis can be made by finding an elevated value for blood amylase. Since penetrating wounds of the abdomen are explored as a general rule, the injury to the pancreas will be disclosed at the time of operation. Cleanly incised wounds of the pancreas may be closed by a few silk sutures. It is wise to drain the area in an effort to prevent a spreading peritonitis (due to the erosive action of the pancreatic secretion) or to prevent hematoma, pseudocyst or abscess, should hemorrhage, leakage or infection occur. In the non-penetrating type of injury, conservative management similar to the Ochsner treatment of peritonitis should be established. By withholding food, the stimulus to pancreatic secretion is decreased. Deflation of the gastrointestinal tract by means of gastric suction or the Miller-Abbott tube is helpful. Surgical drainage is indicated if hematoma, abscess or pseudocyst should develop. These complications may be found weeks or months after the initial injury.

CHRONIC RELAPSING PANCREATITIS

This term has recently been used ([9]) to emphasize the fact that in some patients without associated gall bladder or gastrointestinal disease, attacks of acute pancreatitis recur with free intervals characterized by various degrees of deficiency of either external or internal secretion or both. The disease progresses to produce fibrosis, atrophy, calcification or pseudocysts, and to produce such effects on neighboring structures as obstruction of the duodenum or common duct. The surgical treatment consists of conservative measures such as internal or external drainage of the biliary tract and of pancreatic cysts, removal of pancreatic stones, and gastroenterostomy for duodenal obstruction. More radical procedures occasionally justified are partial or subtotal pancreatectomy.

There has been an increasing interest in the use of sympathetic blocks with novocaine or similar acting drugs in the treatment of chronic relapsing pancreatitis. Attempts to decrease pancreatic secretion either medically by the use of Banthine or surgically by vagotomy and partial gastric resection have been successful in some instances, the benefit attained being secondary to the resulting decrease in gastric secretion.

CHRONIC PANCREATITIS

This is a condition not often diagnosed clinically. However, a symptom complex including dyspepsia, upper abdominal pain, nausea and vomiting, and weakness has been described by some authors. As a rule, the diagnosis is made only

after careful examination has excluded lesions of the gall bladder, stomach, duodenum and other portions of the gastrointestinal tract. It has been said that if an abdominal complaint resembles recurrent appendicitis, gall bladder disease, peptic ulcer or intestinal obstruction, and none of these lesions can be demonstrated, the diagnosis of chronic pancreatitis should be entertained. As a general rule, however, chronic pancreatitis is diagnosed at the operating table and found to be secondary to disease of the biliary tract or to previous attacks of acute pancreatitis. The lesion is represented by fibrosis and enlargement of the gland. Obstructive jaundice may be due to chronic changes in the head of the pancreas, and a mistaken diagnosis of carcinoma may be made. Since no biopsy material is taken to substantiate this clinical impression, the subsequent course must provide the final diagnosis. In those cases surviving cholecystogastrostomy or other internal drainage operations for three or four years, the induration obviously represented benign fibrosis. In general, the treatment of chronic pancreatitis has nothing to do with the pancreas, but is directed toward correction of diseases of the biliary tract. The exceptions occur in those rare instances where pain may indicate resection (see Pancreatic Lithiasis and Calcification.

PANCREATIC LITHIASIS AND CALCIFICATION

Pancreatic calculi, composed chiefly of calcium carbonate, are believed to be the result of infection and stagnation in the ductal system of the pancreas. There are about 250 cases reported ([19]). The chief complaint is colicky pain, located principally in the epigastrium and back, and with radiation along the left costal margin. About one-third of the cases have diabetes or transitory glycosuria. The stools usually remain unchanged. The high calcium content of the pancreatic stones makes them radiopaque, and their linear distribution along the course of the pancreatic duct is highly suggestive. In the evaluation of *x*-ray evidence, it is necessary to rule out biliary or renal calculi and arteriosclerotic plaques in the aorta. In many instances, crepitus may be elicited at operation. The surgical treatment consists of opening the ducts and removing the stones. The parenchyma may be thinned out over the stones, and crepitus between calculi may serve as a guide for blunt or sharp dissection. Drainage of the area is generally employed. If a fistula develops, it does not usually persist for long. Abscess may be associated with stones. Treatment includes drainage of the abscess and removal of the stones.

Pancreatic calcification is extremely rare, there being eighteen cases reported at present ([32]). It is believed to follow repeated attacks of acute pancreatitis, with resulting areas of necrosis, fibrosis and subsequent calcification. The clinical manifestations of diffuse pancreatic calcification are variable. They range from mild, vague, indefinite epigastric discomfort to the very severe type of epigastric pain necessitating morphine, as in the case described by Rienhoff and Baker ([33]). Partial, or rarely total, resection of the pancreas may be necessary for diffuse pancreatic calcification, while attacks on the sympathetic system, such as bilateral vagectomy and sympathectomy as described by Rienhoff and Baker ([33]), or splanchnicectomy as described by DeTakats ([11]), may be effective in relieving pain.

HYPERINSULINISM

Hyperinsulinism is a syndrome first described by Harris ([21]) in 1924. It is characterized by 1) attacks of insulin shock coming on during fasting or in an over-

fatigued state, 2) blood sugar levels of 50 mg. per cent or less and 3) relief by ingestion or administration of dextrose (Whipple's triad). The islet cell tumors, which are most often the cause, are dark red or purplish, due to their vascularity. They are usually 1 to 3 cm. in their greatest dimension, but larger ones are encountered occasionally. They are firmer than the surrounding normal pancreatic tissue and are usually single. Two tumors have been found in 6 to 9 per cent of cases ([11]). As many as five in one patient have been reported. Most islet cell tumors are benign adenomas. However, the diagnosis of islet cell carcinoma has been made in some instances on the basis of the microscopic examination of locally excised tissue, without demonstration of locally invasive or metastatic manifestations. The long survival period in many of these cases justifies reëxamination of the criteria on which the microscopic diagnosis has been made. For that reason, in the absence of demonstrable metastases, local excision of such a tumor encountered at operation is justified without microscopic examination of a frozen section.

Of interest are the reports of neurologic changes associated with eyelet cell tumors. This is not surprising as the cerebral cells are especially sensitive to low levels of blood sugar and patients with the Whipple triad have had in effect a series of insulin shock treatments of mild degree. A manifestation of nonspecific islet cell adenoma of the pancreas is primary peptic ulceration of the jejunum which has been reported recently ([9]).

CARCINOMA OF THE PANCREAS

Carcinoma of the pancreas is the most important surgical lesion of this organ. It constitutes approximately 3 per cent of all deaths due to carcinoma and accounts for 1 in 1000 hospital admissions. The etiology is unknown. The greatest incidence is in the age group from forty to seventy years. It attacks males in the proportion of three to one. Race is without significance.

Adenocarcinoma is almost always the type present, scirrhous carcinoma of the pancreas is extremely rare. About 85 per cent of carcinomas of the pancreas are in the head of the organ with or without involvement of other parts of the gland. Metastases occur by extension to the duodenum, common duct or regional lymph nodes, but they are most common in the liver, being found there twice as frequently as in regional lymph nodes. This is thought to indicate that spread by the blood stream is more important than spread by lymphatics. The peritoneum, the mesentery and the structures in the pelvis are late sites of metastases. Generalized metastases, however, are rare.

Signs and Symptoms.—The "typical picture" (Bard and Pic, 1888) of painless jaundice, distended gall bladder and weight loss is present in only about 20 per cent of cases. Most recent writers agree that inexplicable epigastric pain of a dull, boring, penetrating character is a common early symptom. It is worse when the patient is lying down. It was found in 74 per cent of one series of cases and in from 64 to 100 per cent in other series noted. Weight loss has been stressed by many authors as the second most common symptom. An average loss varying from 5 to 10 pounds per month has been reported and losses even greater than this, amounting to 50 or 60 pounds in a few weeks, have been noted. Jaundice is an outstanding symptom and is due to compression of the common bile duct by the carcinoma in the head of the pancreas. Jaundice has been present in two-thirds to three-quarters of cases in the longer series reported. Dyspepsia of the qualitative

type, nausea and vomiting have been found in about 50 per cent. In the 15 per cent in which the tumor has been found in the body or tail of the pancreas, essentially the same symptoms have been found except for the occurence of jaundice. It is interesting to note that some of these cases have been diagnosed as psychoneuroses because of irritability, headaches and depression, and it is only when jaundice has intervened that the true nature of the patient's difficulty has been realized ([15]). Physical examination reveals evidence of weight loss, jaundice, enlarged liver, tumor or palpable gall bladder in 50 per cent of the cases, and as terminal manifestations there may be ascites and edema. In the examination of the abdomen for the presence of a tumor or of a distended gall bladder, it has been found helpful to give the patient sodium amytal, 2 to 5 grains, intravenously to secure satisfactory relaxation of the abdominal wall. Laboratory findings include those usually present in obstructive jaundice. Blood may be found in the stools if the tumor has invaded the duodenal mucosa, but bleeding from the gastro-intestinal tract can also be due to hypoprothrombinemia.

Because of the difficulty in the diagnosis of carcinoma of the pancreas in the absence of jaundice, it has been suggested that persistent upper abdominal pain extending into the back, worse at night or when lying down, is particularly suggestive of carcinoma of the pancreas, and that exploratory laparotomy is justified in such cases.

Carcinoma of the ampulla of Vater or of the periampullary region is said to occur in younger age groups ([24]). Jaundice occurs before extensive weight loss and other systemic effects. As pointed out by Cattell ([4]), these facts, plus the lower degree of malignancy in carcinoma of the ampulla, make these cases more suitable for resection. Ulceration of the lesion with intestinal bleeding and secondary anemia may occur. The accumulation of follow-up data on patients with pancreatic carcinoma treated by pancreaticoduodenectomy is so discouraging that the radical procedure should be reserved for the most favorable cases, *i.e.*, the early limited lesions in good risk patients. The poor possibility of long-term benefit does not justify the extensive operative procedure in less than the most hopeful circumstances.

Sarcoma of the pancreas is extremely rare, there being 30 cases reported up to 1935. Twenty-nine cases of cystadenocarcinoma and forty-seven cases of benign cystadenoma of the pancreas were reported in 1952 ([40]). Cystadenocarcinoma of the pancreas is also rare. Kennard ([26]), in 1941, collected about 25 cases from the literature, the description of which appeared to justify the diagnosis.

PRE-OPERATIVE PREPARATION OF PATIENTS FOR PANCREATIC SURGERY

Most patients undergoing surgery for pancreatic disease are in older age groups, and accurate evaluation of their cardiovascular and renal status is obligatory. Many have suffered dietary restrictions and in some there has been intestinal bleeding, so that avitaminosis, hypoproteinemia and anemia must be corrected, along with any abnormalities in carbohydrate metabolism. In shorter procedures such as marsupialization of a pseudocyst or drainage of a pancreatic abscess, the general condition of the patient is not taxed as in a long operation. However, resections of the pancreas are formidable procedures and no point should be overlooked in bringing about the maximal improvement in the patient's general condition. Otherwise, his chances of surviving such extensive surgery may be considerably lessened.

In patients about to undergo resection of the pancreas, a non-residue diet, mild saline aperients and the use of a Miller-Abbott tube will collapse the intestinal tract, thus affording maximal room and exposure at operation. Obviously, a factor of great importance is the anesthesia. While continuous spinal anesthesia has been recommended, the choice of anesthetic agent will be secondary to the skill of the anesthesiologist.

Arrangements should be made for transfusion of whatever quantity of blood may be needed for actual replacement or to combat shock. The amount may vary from 500 to 2500 ml. Laboratory facilities for the adequate study of carbohydrate metabolism, both during and after operation, must be provided. This is especially important in subtotal and total pancreatectomy and in surgery for hyperinsulinism.

SURGICAL PROCEDURES FOR PANCREATIC DISEASE

The surgical procedures used in the management of pancreatic disease vary, of course, with the pathology. Drainage is indicated for trauma or for pancreatic abscess. Pseudocysts may be treated by marsupialization or by anastomosis with a segment of the upper gastrointestinal tract. Pancreatic fistulæ may persist after drainage and it may be necessary ultimately to dissect out such fistulous tracts, implanting them in the gastrointestinal tract to prevent continued loss of pancreatic secretion and erosion of the abdominal wall. Obstruction of the common bile duct may necessitate an anastomosis between the biliary and gastrointestinal tracts (see Figs. 485 and 486, Chapter 59, p. 663).

Resections of the pancreas are indicated in carcinoma and other tumors, hyperinsulinism, true cysts, and in some instances of calcification. For carcinomas of the head of the pancreas, resection of the head, the adjacent portion of duodenum and a suitable part of the body of the organ is indicated. For periampullary carcinoma, resection of the head of the pancreas and the adjacent portion of the duodenum is sufficient. Local excision is satisfactory for discrete lesions such as benign tumors, including islet cell tumors, and true cysts of the pancreas. More extensive excision of the body and tail of the organ is indicated in cases of hyperinsulinism in which no islet cell tumor has been found.

Radical resection of the pancreas follows two general plans. In one, the tail and part of the body of the pancreas are removed, together with the spleen. In the other, the duodenum and the head of the pancreas, with or without a portion of the body, are removed. This is indeed a formidable procedure, both from the standpoint of the amount of dissection and from that of the extensive anastomotic procedures necessary to re-establish the continuity of the gastrointestinal and biliary tracts and to provide a suitable connection between the pancreatic duct and the intestinal lumen. Subtotal pancreatectomy, which may be done for hyperinsulinism or for extensive carcinoma, is followed by hyperglycemia and a need for rather large amounts of insulin postoperatively. The carbohydrate metabolism must be studied repeatedly at short intervals in the postoperative period until, after several months, it becomes stabilized. At that time, in may instances, insulin may be withdrawn. The total pancreatectomy ([47]) requires even more rigid study and management of carbohydrate metabolism in the immediate postoperative period but these patients eventually require smaller doses of insulin than some of the patients with subtotal pancreatic resections

The incision may vary with the habitus of the patient. The exposure must be especially generous because of the deep location of the involved structures and the possibility of hemorrhage in dissecting in the region of the portal vein, vena cava and its tributaries. A long right paramedian incision may give adequate exposure to the head of the pancreas, but a lateral extension to the right is often desirable.

In operations on the body or tail of the pancreas, a transverse incision is preferable. For generous exposure of the upper abdomen, an inverted T incision combining a transverse with a vertical midline incision is excellent, but somewhat time-consuming to make and to close. Division of the gastrocolic ligament provides a

Fig. 505.—Kocher's incision to mobilize the duodenum and pancreas

direct approach to the body and tail of the pancreas. If resection of these portions of the gland is to be done, the spleen is removed and the lower margin of that portion of the pancreas is dissected free, this dissection passing in front of the inferior mesenteric vein and exposing the junction of this vein with the splenic vein. The splenic artery is divided and ligated at the point of division of the pancreas. The splenic vein is ligated near its junction with the inferior mesenteric vein. The division of the pancreas should be a wedge-shaped one, leaving a "fish-mouth" defect in the end of the remaining portion of the gland. An adequate closure will be accomplished by bringing together the resultant raw surfaces. The denuded area should be drained.

Resection of the head of the pancreas and adjacent duodenum (duodenopancreatectomy) presents some additional problems. When it has been determined that there are no liver metastases or spread in the region which would make a reasonable hope of cure impossible, resectability must be evaluated as the operation progresses. The dissection begins at the lateral border of the duodenum by incising the peritonium (Kocher's maneuver, Fig. 505). The duodenum and head of the

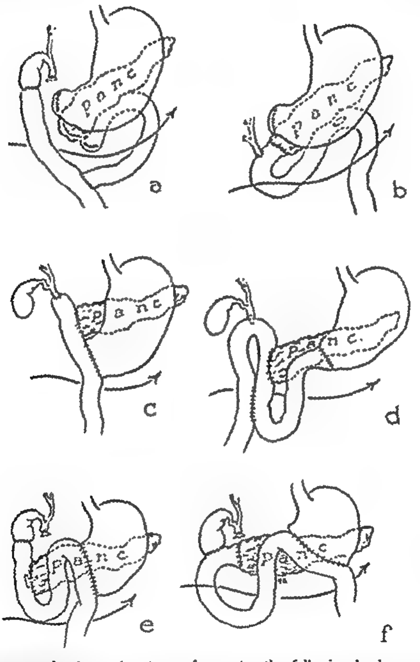

FIG. 506.—Diagrams showing various types of reconstruction following duodenopancreatectomy; a) Whipple[44]; b) Hunt[22]; c) Whipple[44]; d) Cattell[5]; e) Poth[30]; f) Child[4].

pancreas are completely mobilized. The vena cava, the lower end of the common duct and portions of the portal vein and superior mesenteric vessels will thus be exposed. At this time, resectability can be determined. If resection is contraindicated, a palliative procedure such as cholecystogastrostomy may be done. If resection is indicated, the operation is continued by dividing the common duct as low as possible. The uncinate process of the pancreas is freed with particular care to

avoid injuring the superior mesenteric vessels. The stomach is then divided at a convenient level proximal to the pylorus. The head of the pancreas is freed from the portal and splenic veins and the arteries to the region, the inferior and superior pancreatoduodenal vessels, may be divided and ligated. The bowel is now divided in the region of the ligament of Treitz and the pancreas is cut across. The series of anastomoses is then completed by a choledochojejunostomy, a pancreaticojejunostomy and a gastrojejunostomy (Fig. 506). It is wise to drain the area which has been denuded of peritoneal covering.

The choice of a one or two-stage pancreatoduodenal resection is based upon the general condition of the patient and the degree of jaundice present. Cattell (4) has emphasized the value of preliminary internal drainage of the biliary system in patients with severe jaundice. He further points out that in the first stage of a two-stage procedure, dissection in the region of the duodenum or the common duct should not be done, so as to avoid the occurrence of adhesions which would make the second stage much more difficult.

The fundamental principles as outlined by Orr (32) include 1) removal of sufficient tissue about the tumor to include any local infiltration or regional lymph nodes, 2) use of the common duct when possible, instead of the gall bladder, for anastomosis to avoid leakage from the ligated stump of the common duct, 3) gastrojejunostomy distal to the other anastomosis to avoid infection of the biliary tract and pancreatic parenchyma by regurgitation, 4) anastomosis of the pancreatic duct and intestinal tract to avoid pancreatic fistulæ and to restore pancreatic secretions to the intestinal tract. The one-stage operations of Whipple (43), Poth (36), Child (6) and Hunt (23) and the one- and two-stage procedures described by Cattell (4) accomplish these objectives (Fig. 506). The anastomoses in Cattell's plan are all anticolic. In the one-stage procedure, the common bile duct is used for the anastomosis. In the first stage of Cattell's two-stage procedure, a cholecystojejunostomy is done to avoid manipulations and resultant adhesions in the area about the common duct which is to be dissected at the second stage.

One of the principles in the operation, whether one-stage or two-stage, which has prompted considerable discussion is the avoidance of ascending cholangitis by allowing the gastric contents to enter the gastrointestinal tract distal to the intestinal biliary connection. The Roux-Y procedure, as advocated by Whipple (43), Cole and Reynolds (8) and others (30), deserves special mention. There is a growing conviction that pancreaticointestinal anastomosis will improve the patient's nutritional status, but perhaps more important, as Pearse (35) points out, it will avoid to a large measure troublesome fistulæ which have occurred frequently following ligation of the pancreatic duct.

Recently, Owens (34) has called attention to the problem of peptic ulcer following pancreatectomy. This tendency towards ulceration in the absence of pancreatic secretion furnishes an additional reason for anastomosing the pancreatic duct to some portion of the upper intestinal tract.

The postoperative care must be zealously managed to avoid complications where possible and to cope with them, should they arise. Adequate fluid and blood replacement, gastrointestinal decompression, maintenance of a satisfactory nutritional state, regulation of carbohydrate metabolism, and chemotherapy and antibiotics are the major points. The status of the patient's general health pre-operatively will suggest special cautions to avoid cardiovascular, renal or pulmonary complications.

The results of pancreatoduodenectomy for carcinoma have prompted a difference of opinion concerning the performance of a procedure which has an operative mortality of 10 to 20 per cent in an effort to attain the current five-year survival rate of approximately one per cent. Nevertheless, it is agreed that radical surgery offers the only possibility of cure.

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The choice of a one or two-stage pancreatoduodenal resection is based upon the general condition of the patient and the degree of jaundice present. Cattell (5) has emphasized the value of preliminary internal drainage of the biliary system in patients with severe jaundice. He further points out that in the first stage of a two-stage procedure, dissection in the region of the duodenum or the common duct should not be done, so as to avoid the occurrence of adhesions which would make the second stage much more difficult.

The fundamental principles as outlined by Orr (33) include 1) removal of sufficient tissue about the tumor to include any local infiltration or regional lymph nodes, 2) use of the common duct when possible, instead of the gall bladder, for anastomosis to avoid leakage from the ligated stump of the common duct, 3) gastrojejunostomy distal to the other anastomosis to avoid infection of the biliary tract and pancreatic parenchyma by regurgitation, 4) anastomosis of the pancreatic duct and intestinal tract to avoid pancreatic fistulæ and to restore pancreatic secretions to the intestinal tract. The one-stage operations of Whipple (43), Poth (36), Child (6) and Hunt (23) and the one- and two-stage procedures described by Cattell (5) accomplish these objectives (Fig. 506). The anastomoses in Cattell's plan are all anticolic. In the one-stage procedure, the common bile duct is used for the anastomosis. In the first stage of Cattell's two-stage procedure, a cholecystojejunostomy is done to avoid manipulations and resultant adhesions in the area about the common duct which is to be dissected at the second stage.

One of the principles in the operation, whether one-stage or two-stage, which has prompted considerable discussion is the avoidance of ascending cholangitis by allowing the gastric contents to enter the gastrointestinal tract distal to the intestinal biliary connection. The Roux-Y procedure, as advocated by Whipple (43), Cole and Reynolds (8) and others (30), deserves special mention. There is a growing conviction that pancreaticointestinal anastomosis will improve the patient's nutritional status, but perhaps more important, as Pearse (35) points out, it will avoid to a large measure troublesome fistulæ which have occurred frequently following ligation of the pancreatic duct.

Recently, Owens (34) has called attention to the problem of peptic ulcer following pancreatectomy. This tendency towards ulceration in the absence of pancreatic secretion furnishes an additional reason for anastomosing the pancreatic duct to some portion of the upper intestinal tract.

The postoperative care must be zealously managed to avoid complications where possible and to cope with them, should they arise. Adequate fluid and blood replacement, gastrointestinal decompression, maintenance of a satisfactory nutritional state, regulation of carbohydrate metabolism, and chemotherapy and antibiotics are the major points. The status of the patient's general health pre-operatively will suggest special cautions to avoid cardiovascular, renal or pulmonary complications.

The results of pancreatoduodenectomy for carcinoma have prompted a difference of opinion concerning the performance of a procedure which has an operative mortality of 10 to 20 per cent in an effort to attain the current five-year survival rate of approximately one per cent. Nevertheless, it is agreed that radical surgery offers the only possibility of cure.

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CHAPTER 62

SURGERY OF THE SPLEEN

By Steven O. Schwartz

AND

TECHNIQUE OF SPLENECTOMY

By Robert L. Schmitz

HISTORICAL BACKGROUND

The history of splenectomy reaches into antiquity. It is said to have been performed for marathon runners to improve their endurance. The first splenectomy in a human being is recorded as having taken place in 1549, executed by Fioravanti. His patient died. In 1680 Zambeccari's splenectomy in the dog was successful, but the first known successful splenectomy of modern times was not until almost 200 years later, in 1865, with Spencer Wells as the surgeon.

By 1887, 63 splenectomies in the human being had been reported. In 1888 Spencer Wells performed the first splenectomy for congenital hemolytic jaundice. His patient was well 45 years later. The first splenectomy for Gaucher's disease was performed in 1896 by Picou and Raymond. By 1898, 274 splenectomies with 170 recoveries had been reported. Kaznelson did the first splenectomy for thrombocytopenic purpura in 1916.

EMBRYOLOGY

The spleen first appears at the beginning of the fifth week of fetal life, as a localized growth in the mesoderm of the mesogastrium. It develops in the dorsal mesogastrium of the embryo. This is important for an understanding of the location of accessory spleens, to be discussed hereinafter.

ANATOMIC CONSIDERATIONS

The spleen lies under the anterior lateral aspect of the left leaf of the diaphragm. Normally it weighs between 100 and 250 gm. with an average weight of about 150 gm. It is somewhat larger in the male than in the female and somewhat smaller in the Negro than in the Caucasian. It is supplied by the splenic artery which is the largest branch of the celiac axis, and is drained by the splenic vein which is the largest tributary of the portal vein. It is made up of a supporting connective tissue whose constituents are the splenic capsule, the trabeculæ, and the reticulum framework. The vascular system is extremely complex and the nature of the circulation is uncertain, both the "closed" and "open" circulation schools of thought having large groups of supporters. Whether or not the circulatory system of the spleen is open or closed, there is at least working agreement that circulation of the blood in the spleen is extremely sluggish. This is physiologically

important, because sluggish circulation leads to erythrostasis, and erythrostasis in turn results in physico-chemical as well as morphologic changes in the red cells which permit their destruction by mechanical, chemical or hormonal means.

The splenic pulp consists of the white and the red pulp. The former is made up of malpighian bodies (splenic follicles) which consist of lymphatic tissue surrounding the splenic arterioles and other lymphoid tissue. The red pulp is composed of the circulating blood cells, reticulo-endothelial cells, reticular fibers and lymphocytes.

PHYSIOLOGY

The spleen has many functions; but fortunately its activities are shared by other organs; therefore, it is not essential to life and its removal does not lead to serious disability or handicap.

Ordinarily the spleen's functions are fivefold: The participation of the spleen (1) in blood formation; (2) in the body defense mechanism; (3) in the destruction of the red blood cells and perhaps also in the destruction of the white blood cells and platelets; (4) importantly in the control of blood formation and delivery of cells from the marrow and probably from the lymphoid organs; (5) in its reservoir function in cases in which sudden demand is made on the spleen for the delivery of needed red cells. Some exposition of these functions is in order:

1. *Participation of the Spleen in Blood Formation.*—This function is especially prominent in the embryo where the spleen is active in hemopoiesis; however; in the adult it normally participates in blood formation only in that it produces lymphocytes and monocytes. Under abnormal conditions and under unusual physiologic stresses, the spleen may revert to an embryonal hemopoietic function and produce all types of blood cells.

2. *Body Defense Mechanism.*—The spleen produces antibodies and is of primary importance in recovery from radiation injury.

3. *Destruction of Red Blood Cells; Perhaps Destruction of White Cells and Platelets.*—The sluggish circulation in the spleen which results in stasis of the cells alters the physical characteristics of the red cells, so that they become greater in thickness and tend to be spherical in shape, especially as the cells get older. Whether these physical changes in the cells result in their destruction in the spleen by virtue of sequestration, phagocytosis or other mechanisms is controversial.

4. *Control of Blood Formation and Delivery from Marrow.* The control of blood formation and delivery from the marrow and probably from the lymphoid organs is probably as important a function in the adult as any in the development of surgical conditions necessitating splenectomy. This function will be further discussed under the topic of *Hypersplenism.* There is evidence that some of the function resulting in the formation and delivery of cells from the marrow is hormonal in nature and acts by influencing the marrow and lymphoid tissues.

5. *Reservoir Function.*—When sudden demand is made on the spleen for the delivery of needed red cells, the reservoir function is important. The reservoir function is under autonomic control and is influenced by exercise, excitement, and anoxia—all of which can produce a contraction of the spleen to make possible utilization of the cells normally contained in the spleen, in the form of an autotransfusion. The potential significance of this appears in patients who previously have been splenectomized and require additional surgery, for such patients, without

the reservoir function of the spleen available to them, might readily go into shock during or following operations.

SPLENOMEGALY

Enlargement of the spleen may be the result of infection, congestion, infiltration, metaplasia, primary tumors and cysts, or proliferation of reticulum tissues intrinsic to the spleen. Enlargement of the spleen also takes place in the various hemolytic anemias.

The acute infectious diseases best known to produce splenomegaly are infectious mononucleosis, malaria, typhoid, and measles. The subacute infectious diseases are bacterial endocarditis, malaria, brucellosis, and histoplasmosis. Chronic infectious diseases are malaria, kala-azar, abscesses, tuberculosis, and syphilis. Occasionally in diseases whose etiology is unknown, but which may be related to the chronic infections such as lupus erythematosus, sarcoid of Boeck, and rheumatoid arthritis, there is an enlargement of the spleen. The amyloidosis that occasionally accompanies or follows these infections may also be an etiologic factor in the development of splenomegaly.

The second group of diseases characterized by splenomegaly are those resulting from congestion of the spleen because of partial or complete obstruction of the splenic vein. This may be due to an obstruction of the splenic vein itself, an obstruction of the splenic vein caused by a portal vein obstruction, or hypertension of a splenic vein from obstruction in the liver, as in cirrhosis.

Hemolytic anemias are also accompanied by enlargement of the spleen. The most prominent splenomegalies are those accompanying congenital spherocytosis. Moderate splenomegalies accompany acquired hemolytic anemias and the various hemoglobinurias. Sickle cell anemia is accompanied by splenomegaly only during childhood. After the age of twelve it is rare to find splenomegaly in sickle cell anemia; on the contrary, the spleen in the adult may become so small as a result of repeated infarctions as to become difficult to find on post-mortem examination. Some of the more unusual abnormal hemoglobinopathies, such as hemoglobin C disease, hemoglobin D disease, and hemoglobin E disease, both in their homozygous form and when accompanying either sickle cell disease or when co-existing with sickle cell hemoglobin or Mediterranean hemoglobin, are also characterized by splenomegalies. Splenomegaly which is part and parcel of Mediterranean anemia is variable, the spleen size being roughly in proportion to the severity of the disease. The largest splenomegalies in this condition are seen in children having thalassemia major, otherwise known as Cooley's disease or severe Mediterranean anemia. The milder forms of the disease may be accompanied by minimal splenomegalies and insignificant hematologic abnormalities.

Polycythemia vera is most frequently accompanied by splenomegaly and this may reach extreme proportions. Myeloid metaplasia of the spleen is a term given to a disease characterized by fibrosis of the marrow and abnormal hemopoiesis in the spleen. The various leukemias and lymphomas as well as Hodgkin's disease often exhibit splenomegaly. The lipoid dystrophies, of which Gaucher's disease, Niemann-Pick disease, Hand-Schüller-Christian disease, and the nonlipoid variant known as Letterer-Siwe disease, are found in some instances in conjunction with large spleens. Primary tumors of the spleen and cysts of the spleen are not common but must be included in the differential diagnosis of splenic tumors.

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CHAPTER 62

SURGERY OF THE SPLEEN

By Steven O. Schwartz

AND

TECHNIQUE OF SPLENECTOMY

By Robert L. Schmitz

HISTORICAL BACKGROUND

The history of splenectomy reaches into antiquity. It is said to have been performed for marathon runners to improve their endurance. The first splenectomy in a human being is recorded as having taken place in 1549, executed by Fioravanti. His patient died. In 1680 Zambeccari's splenectomy in the dog was successful, but the first known successful splenectomy of modern times was not until almost 200 years later, in 1865, with Spencer Wells as the surgeon.

By 1887, 63 splenectomies in the human being had been reported. In 1888 Spencer Wells performed the first splenectomy for congenital hemolytic jaundice. His patient was well 45 years later. The first splenectomy for Gaucher's disease was performed in 1896 by Picou and Raymond. By 1898, 274 splenectomies with 170 recoveries had been reported. Kaznelson did the first splenectomy for thrombocytopenic purpura in 1916.

EMBRYOLOGY

The spleen first appears at the beginning of the fifth week of fetal life, as a localized growth in the mesoderm of the mesogastrium. It develops in the dorsal mesogastrium of the embryo. This is important for an understanding of the location of accessory spleens, to be discussed hereinafter.

ANATOMIC CONSIDERATIONS

The spleen lies under the anterior lateral aspect of the left leaf of the diaphragm. Normally it weighs between 100 and 250 gm. with an average weight of about 150 gm. It is somewhat larger in the male than in the female and somewhat smaller in the Negro than in the Caucasian. It is supplied by the splenic artery which is the largest branch of the celiac axis, and is drained by the splenic vein which is the largest tributary of the portal vein. It is made up of a supporting connective tissue whose constituents are the splenic capsule, the trabeculæ, and the reticulum framework. The vascular system is extremely complex and the nature of the circulation is uncertain, both the "closed" and "open" circulation schools of thought having large groups of supporters. Whether or not the circulatory system of the spleen is open or closed, there is at least working agreement that circulation of the blood in the spleen is extremely sluggish. This is physiologically

important, because sluggish circulation leads to erythrostasis, and erythrostasis in turn results in physico-chemical as well as morphologic changes in the red cells which permit their destruction by mechanical, chemical or hormonal means.

The splenic pulp consists of the white and the red pulp. The former is made up of malpighian bodies (splenic follicles) which consist of lymphatic tissue surrounding the splenic arterioles and other lymphoid tissue. The red pulp is composed of the circulating blood cells, reticulo-endothelial cells, reticular fibers and lymphocytes.

PHYSIOLOGY

The spleen has many functions; but fortunately its activities are shared by other organs; therefore, it is not essential to life and its removal does not lead to serious disability or handicap.

Ordinarily the spleen's functions are fivefold: The participation of the spleen (1) in blood formation; (2) in the body defense mechanism; (3) in the destruction of the red blood cells and perhaps also in the destruction of the white blood cells and platelets; (4) importantly in the control of blood formation and delivery of cells from the marrow and probably from the lymphoid organs; (5) in its reservoir function in cases in which sudden demand is made on the spleen for the delivery of needed red cells. Some exposition of these functions is in order:

1. *Participation of the Spleen in Blood Formation.*—This function is especially prominent in the embryo where the spleen is active in hemopoiesis; however; in the adult it normally participates in blood formation only in that it produces lymphocytes and monocytes. Under abnormal conditions and under unusual physiologic stresses, the spleen may revert to an embryonal hemopoietic function and produce all types of blood cells.

2. *Body Defense Mechanism.*—The spleen produces antibodies and is of primary importance in recovery from radiation injury.

3. *Destruction of Red Blood Cells; Perhaps Destruction of White Cells and Platelets.*—The sluggish circulation in the spleen which results in stasis of the cells alters the physical characteristics of the red cells, so that they become greater in thickness and tend to be spherical in shape, especially as the cells get older. Whether these physical changes in the cells result in their destruction in the spleen by virtue of sequestration, phagocytosis or other mechanisms is controversial.

4. *Control of Blood Formation and Delivery from Marrow.* The control of blood formation and delivery from the marrow and probably from the lymphoid organs is probably as important a function in the adult as any in the development of surgical conditions necessitating splenectomy. This function will be further discussed under the topic of *Hypersplenism.* There is evidence that some of the function resulting in the formation and delivery of cells from the marrow is hormonal in nature and acts by influencing the marrow and lymphoid tissues.

5. *Reservoir Function.*—When sudden demand is made on the spleen for the delivery of needed red cells, the reservoir function is important. The reservoir function is under autonomic control and is influenced by exercise, excitement, and anoxia—all of which can produce a contraction of the spleen to make possible utilization of the cells normally contained in the spleen, in the form of an autotransfusion. The potential significance of this appears in patients who previously have been splenectomized and require additional surgery, for such patients, without

the reservoir function of the spleen available to them, might readily go into shock during or following operations.

SPLENOMEGALY

Enlargement of the spleen may be the result of infection, congestion, infiltration, metaplasia, primary tumors and cysts, or proliferation of reticulum tissues intrinsic to the spleen. Enlargement of the spleen also takes place in the various hemolytic anemias.

The acute infectious diseases best known to produce splenomegaly are infectious mononucleosis, malaria, typhoid, and measles. The subacute infectious diseases are bacterial endocarditis, malaria, brucellosis, and histoplasmosis. Chronic infectious diseases are malaria, kala-azar, abscesses, tuberculosis, and syphilis. Occasionally in diseases whose etiology is unknown, but which may be related to the chronic infections such as lupus erythematosus, sarcoid of Boeck, and rheumatoid arthritis, there is an enlargement of the spleen. The amyloidosis that occasionally accompanies or follows these infections may also be an etiologic factor in the development of splenomegaly.

The second group of diseases characterized by splenomegaly are those resulting from congestion of the spleen because of partial or complete obstruction of the splenic vein. This may be due to an obstruction of the splenic vein itself, an obstruction of the splenic vein caused by a portal vein obstruction, or hypertension of a splenic vein from obstruction in the liver, as in cirrhosis.

Hemolytic anemias are also accompanied by enlargement of the spleen. The most prominent splenomegalies are those accompanying congenital spherocytosis. Moderate splenomegalies accompany acquired hemolytic anemias and the various hemoglobinurias. Sickle cell anemia is accompanied by splenomegaly only during childhood. After the age of twelve it is rare to find splenomegaly in sickle cell anemia; on the contrary, the spleen in the adult may become so small as a result of repeated infarctions as to become difficult to find on post-mortem examination. Some of the more unusual abnormal hemoglobinopathies, such as hemoglobin C disease, hemoglobin D disease, and hemoglobin E disease, both in their homozygous form and when accompanying either sickle cell disease or when co-existing with sickle cell hemoglobin or Mediterranean hemoglobin, are also characterized by splenomegalies. Splenomegaly which is part and parcel of Mediterranean anemia is variable, the spleen size being roughly in proportion to the severity of the disease. The largest splenomegalies in this condition are seen in children having thalassemia major, otherwise known as Cooley's disease or severe Mediterranean anemia. The milder forms of the disease may be accompanied by minimal splenomegalies and insignificant hematologic abnormalities.

Polycythemia vera is most frequently accompanied by splenomegaly and this may reach extreme proportions. Myeloid metaplasia of the spleen is a term given to a disease characterized by fibrosis of the marrow and abnormal hemopoiesis in the spleen. The various leukemias and lymphomas as well as Hodgkin's disease often exhibit splenomegaly. The lipoid dystrophies, of which Gaucher's disease, Niemann-Pick disease, Hand-Schüller-Christian disease, and the nonlipoid variant known as Letterer-Siwe disease, are found in some instances in conjunction with large spleens. Primary tumors of the spleen and cysts of the spleen are not common but must be included in the differential diagnosis of splenic tumors.

bleeding tendency characterized by petechiæ, ecchymoses and bleeding from the various body orifices. The blood is characterized by thrombocytopenia with or without leukopenia and the absence of anemia. Physical findings are limited to evidences of bleeding. The spleen is rarely palpable in this condition; when it is felt, one should look for secondary thrombocytopenia resulting from splenic involvement by a primary or secondary process. Unless the process is extremely fulminant in idiopathic thrombocytopenic purpura, therapy should be conservative, consisting of intravenous administration of ACTH and oral administration of cortisone. If the patient fails to respond to this regimen or fails to maintain response on discontinuance of this medication, splenectomy is indicated.

Splenectomy is curative in approximately 60 per cent of the cases. About 20 per cent of the patients are made no better by splenectomy, and about 20 per cent are clinically improved but have a residual persistent thrombocytopenia. Unfortunately there is no way of predicting in advance which patients will respond adequately to splenectomy and which will fail. The marrow in all instances is similar and the histologic changes in the spleen, which are characterized by some reticulo-endothelial hyperplasia, are similar in the two conditions.

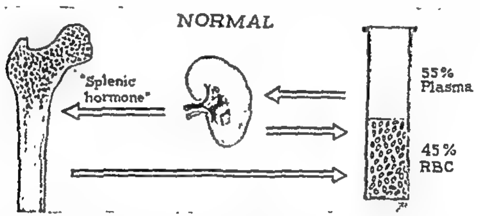

FIG. 507.—The spleen produces a hormone which regulates normal maturation and release of cells from marrow. The blood like a mirror reflects what happens in the spleen and marrow, and acts as a vehicle to carry "splenic hormone" and formed elements.

The third indication for splenectomy is the presence of *leukopenia with neutropenia*. Although a number of patients have uneventful clinical courses even though there is a moderate to marked leukopenia, an occasional patient is encountered in whom leukopenia results in frequent infections, sometimes accompanied by angina. Such patients almost always have concomitant splenomegaly and not infrequently have joint manifestations. The triad of joint pain, leukopenia and splenomegaly is often referred to as Felty's syndrome. Some of these patients are benefited by splenectomy. One has to rule out other diseases, such as lupus erythematosus, which may simulate this syndrome.

Although anemia, thrombocytopenia and leukopenia may occur singly, the cytopenias may occur in combinations. When they occur as a result of an involvement of the spleen, the condition is referred to as *hypersplenism*.

Hypersplenism.—The exact mechanism of hypersplenism remains unelucidated. Banti, as early as 1910, Kaznelson (1916) Wiseman and Doan (1939) ascribed the effect of hypersplenism to a phagocytic function of the spleen. A hormonal effect *acting indirectly on the marrow was postulated by Isaacs in 1912* and his followers have included Frank (1915), Minot (1917) and Dameshek (1941).

Hypersplenism is a term implying a functional defect in the spleen manifesting itself by its adverse effect on marrow function. To fulfill the diagnostic criteria of hypersplenism, one must demonstrate a quantitative diminution in one or more of the formed elements in the peripheral blood, an enlargement of the spleen, and a qualitatively and quantitatively adequate normal marrow. Hypersplenism should always be considered in the presence of splenomegaly and conversely splenic enlargement is always present in hypersplenism. The size of the spleen however, may vary from a barely palpable 250 gm. spleen to tremendous splenomegalies of from 2500 to 5000 gm. The precursors of the cells which are scant in the blood are usually

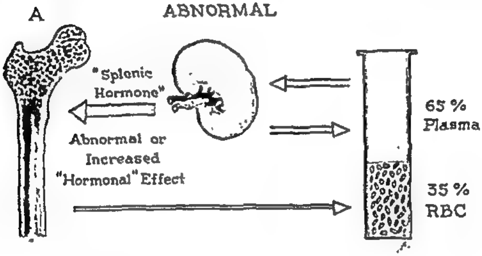

Fig. 508 —The "splenic hormone" acts as inhibitor of normal cell maturation and release from marrow.

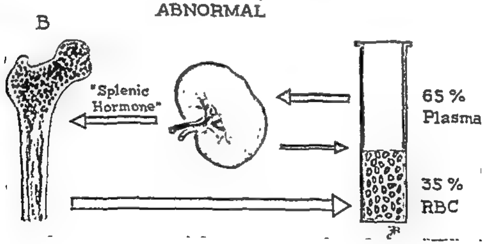

Fig 509.—"Sequestration" of formed elements during circulation in the spleen.

found in great abundance in the hypercellular marrow. In some instances no change in the marrow, except hyperplasia, is demonstrable, whereas in others the same primary disease that involves the spleen may be found in the marrow. Examples of these are the granulomata of tuberculosis, sarcoidosis, histoplasmosis and Hodgkin's disease; the reticulo-endothelial proliferation as seen in Gaucher's disease; the various lymphomatous involvements, such as giant follicle lymphoma, lymphosarcoma and lymphatic leukemia, and finally myelogenous leukemia. Splenic enlargement, on the other hand, may be due to any of the causes already mentioned

as being responsible for splenomegaly. The various mechanisms in hypersplenism are illustrated herewith:

The normal spleen may best be thought of as producing a hormone that regulates normal maturation and release of cells from marrow, and as removing effete cells (Fig. 507). The normal blood reflects the interplay of effects of the spleen and the marrow, and acts as a vehicle to carry the splenic hormone and the formed elements.

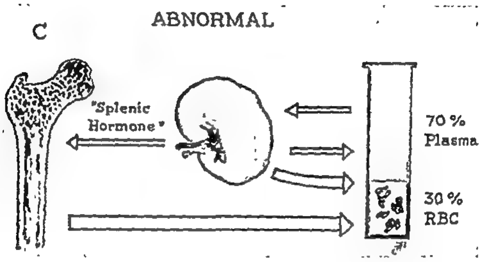

FIG. 510.—Formation of cell lysins or agglutinins

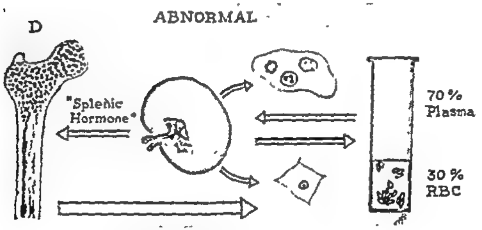

FIG. 511.—Elaboration of R-E stimulating substance which, in turn, leads to phagocytosis of cells, or liberation of agglutinating or lytic substances.

Some possible explanations of *hypersplenism* may be outlined:

1. When there is an abnormal or increased hormonal effect, the spleen acts as an inhibitor of normal cell maturation and release from marrow (Fig. 508). The blood will reflect this inhibition in showing fewer red cells, or white cells, or platelets, or various combinations of these cytopenias.

2. The blood will reflect the same changes when the abnormal condition of the spleen is that of sequestration of formed elements during circulation in the spleen (Fig. 509).

3. A third abnormal situation may occur if the spleen forms abnormal cell lysins or agglutinins. Under this circumstance, disintegration of cells occurs in the circulating blood after their release from the marrow (Fig. 510).

4. A fourth abnormal condition may be caused by an elaboration by the spleen of a reticulo-endothelial stimulating substance which in turn leads to either phagocytosis of cells or liberation of agglutinating or lytic substances (Fig. 511).

Another indication for splenectomy is *congestive splenomegaly*. This may be due to splenic vein thrombosis, portal vein thrombosis, or intrahepatic obstruction from cirrhosis. Normally venous pressure in the splenic vein is between 100 and 120 mm. of water; in congestive splenomegaly this pressure may rise to 250 to 500 mm. of water. Splenectomy in cases of congestive splenomegaly will in most instances relieve the symptom of hypersplenism. As a definitive operative procedure, however, it is not recommended in itself but should be done in conjunction with a portacaval or splenorenal shunt. Occasionally when the patient is extremely ill, manifestations of hypersplenism are severe and the risk of splenectomy or shunt procedure is too great; then, ligation of the splenic artery may be performed with some success.

In cases of *secondary splenomegaly* the indications for splenectomy are two: (1) enlargement of the spleen to a point where it is mechanically embarrassing, or (2) because of severe hypersplenic manifestations. In these cases, even though the underlying disease remains unaltered, the manifestations may be sufficiently influenced so as to afford the patient measurable improvement in health and well-being. In all of these cases, however, before splenectomy is undertaken one has to weigh the probabilities of success carefully and with a full knowledge of the risks imposed. In all cases in which splenectomy is considered, the history, physical examination, blood and marrow findings must be assayed with care. Above all, it is important in each instance to rule out leukemias, lymphomas, the various hemolytic anemias not expected to respond to splenectomy, and myeloid metaplasia.

Primary neoplasms of the spleen are relatively rare. They consist of hemangiomas, lymphangiomas, primary sarcomas, primary lymphosarcomas, and cysts. The cysts are usually traumatic in origin.

ACCESSORY SPLEENS

Accessory spleens are found in approximately 10 per cent of all adult autopsies. In children under seven, accessory spleens are found in about 25 per cent; with advancing age, atrophy is gradual, and by the age of ten, the accessory spleens are found in only approximately 15 per cent of the cases. The accessory spleens are found in the following locations in the order of their frequency: (1) the hilus of the spleen; (2) the gastrosplenic ligament; (3) splenocolic ligament; (4) pancreaticosplenic ligament; (5) greater omentum; (6) along the vessels of the spleen; (7) the gastrohepatic omentum. They may, however, be found in other locations as well and have been reported found in the inguinal canal, in the scrotum, elsewhere in the peritoneum, and are even found embedded, rarely, in the liver and the pancreas. They take on special importance because there is suggested evidence that when the spleen is structurally or functionally deficient, a compensatory hyperplasia of accessory splenic tissue may take place. In connection with the consideration of accessory spleens, mention should be made of *splenosis*. Splenosis is the term applied to the enlargement of splenic particles resulting from implants of splenic tissue in the peritoneal cavity. These may occur either as the consequence of surgical removal of the spleen when the spleen has been torn during removal, or as a result of implants when the spleen has been ruptured by trauma. The number of splenic

particles thus implanted may be great, as many as 1000 having been reported after a gunshot wound with splenic injury.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of splenic tumors includes both benign and malignant tumors of the kidney, such as kidney cysts, hydronephrosis, polycystic kidneys, and carcinoma of the kidneys. Tumors of the pancreas and pancreatic cysts may be mistaken for splenic tumors. Retroperitoneal sarcomas may simulate splenic tumors. Carcinoma of the splenic flexure may give the appearance of a splenic tumor. *Carcinoma and sarcoma of the stomach and occasionally a trichobezoar* of the stomach may give identical physical findings. These other conditions may be differentiated from splenic tumors in one of two fashions: (1) No explanation for a splenomegaly can be found or (2) the signs and symptoms are not explainable on the basis of splenic enlargement. In some of the obscure cases, it is occasionally necessary to resort to roentgenographic examination of the gastrointestinal and urinary tract in order to ascertain the nature of the mass. *Even with the most* rigid conditions of control, meticulous care and preoperative evaluation, an occasional splenic tumor is found to be due to something other than that anticipated on exploration.

PREOPERATIVE AND POSTOPERATIVE CONSIDERATIONS

The most important of all preoperative considerations in splenectomy is the establishment of a precise diagnosis by careful clinical, hematologic and marrow studies. Because of the difficulty accompanying splenectomy and because of the

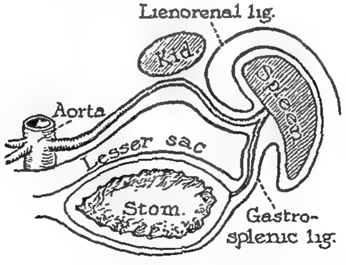

FIG 512 —Anatomical relationship of the spleen.

frequency with which anemia accompanies the disease for which splenectomy is performed, preoperative and operative transfusions are almost invariably essential. Exceptions to this are in thrombocytopenic purpura, in which anemia is an infrequent complication, and in the hemolytic anemias, in which transfusions may aggravate the hemolytic process It is especially important in preoperative consideration to rule out liver disease and pulmonary disease.

The principles applying to the operative procedure are the following: First, it is imperative that splenectomy be performed with as much speed as possible; second, a diligent and thorough search should be made for accessory spleens; third, it is important that the capsule remain intact lest splenic implants (splenosis) follow; fourth, if the spleen is large the spleen may be injected with epinephrine to shrink it, or epinephrine may be put into the splenic artery. As soon as the splenic pedicle has been isolated, the splenic artery should be ligated in order to reduce the size of the spleen, so that its removal may be made easier and also in order that the patient may receive the blood contained in the organ. Ligation of the splenic vein should always be left for the last procedure.

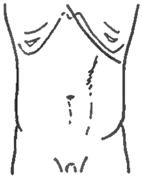

Fig. 513.—Splenectomy. The suggested incision.

Postoperatively a left lower lobe atelectasis complicated by pneumonia is the rule rather than the exception. Because of this, antibiotic therapy is prophylactically advisable. Because of the further complication of portal vein thrombosis and the usual increase in the thrombocytes after splenectomy, early ambulation should be practiced. Routine use of anticoagulants, such as heparin and dicumarol, are not indicated except when thrombotic or embolic manifestations are present, but they may have to be used also in the patient in whom platelets rise to extraordinarily high levels or in those patients in whom early ambulation is not possible.

TECHNIQUE OF SPLENECTOMY

By Robert L. Schmitz

Anatomy.—The spleen is suspended high in the anterolateral fossa of the left upper abdomen under cover of the lower thoracic cage. The outer surface matches the curves of the diaphragmatic reflection, to which it is attached by a band of adhesions of varying proportion and vascularity. The inner surface matches the curves of the stomach to which it is attached by the gastrosplenic ligament which contains the vasa brevia and, in its inferior edge, the left gastroepiploic vessels (Fig. 514). In the posterolateral wall of the lesser omental bursa, the hilus of the spleen embraces the tail of the pancreas, from along the superior margin of which run the splenic artery and vein to form the splenic pedicle (Fig. 512). The left kidney lies behind and somewhat below the spleen; from off of it the retroperitoneum is reflected onto the posterior surface of the splenic pedicle to form the lienorenal ligament (Fig. 512). Finally, paralleling the phrenocolic ligament, there are

a few fibrous strands between the lower pole of the spleen and the splenic flexure of the colon called the splenocolic ligament.

General Considerations.—Removal of the spleen entails the division of its various ligaments and the pedicle (Fig. 512). When the spleen is normal, for example, when it is removed incidental to another operation, there is no difficulty. However, when the spleen is markedly enlarged or when the collateral circulation about the spleen is very rich, splenectomy can become a formidable procedure. Under such circumstances, it may even be wise to approach the spleen through

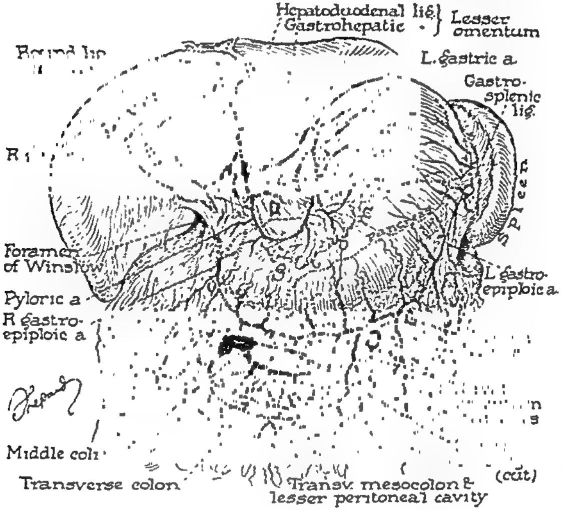

FIG. 514.—Anatomical relationship of the spleen to the stomach.

the left chest and diaphragm to have better access to the pedicle and to the rich collaterals in the diaphragmatic attachments and the upper gastrosplenic ligament, but ordinarily, a left upper abdominal incision is used.

When the spleen is ruptured, from external force or during an operative procedure, it may be necessary to abandon the niceties of the usual orderly routine for an improvised attack which will rapidly deliver the spleen and secure the pedicle.

In diseases of the spleen accompanied by portal hypertension it may be indicated to join the portal and caval venous systems to decrease the portal pressure with its harmful sequellæ. In such instances, when the spleen is removed a splenorenal shunt can be established by anastomosing the cut end of the splenic vein to the side of the left renal vein.

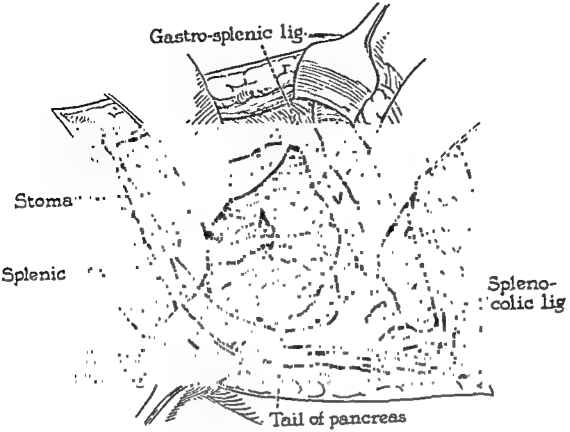

FIG. 515.—Splenectomy. Exposure of pedicle; artery ligated in continuity.

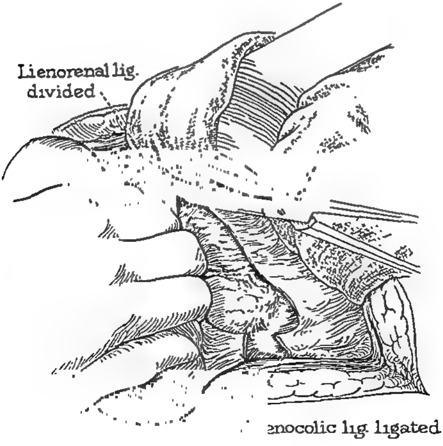

FIG 516—Splenectomy. Mobilization of the spleen posteriorly.

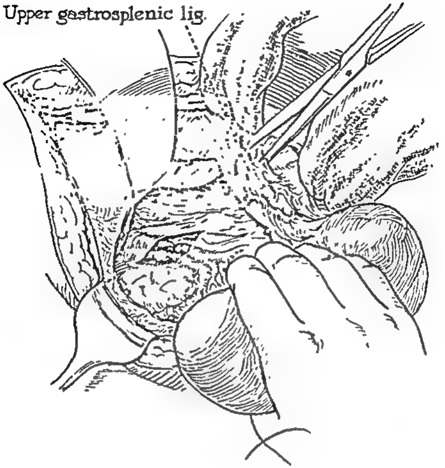

FIG 517 —Splenectomy. Mobilization of the spleen from the stomach and superiorly.

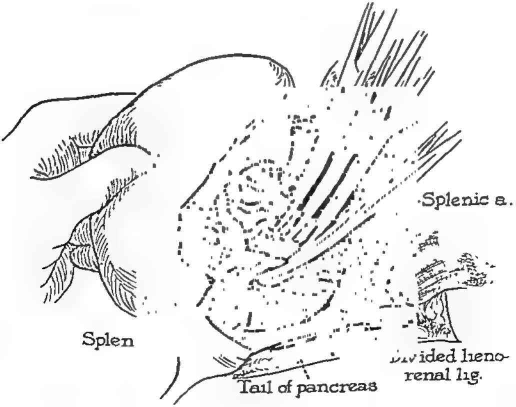

FIG 518 —Splenectomy Securing the pedicle.

Technique.—A left oblique subcostal incision is usually very satisfactory (Fig. 513).

The left third of the gastrocolic ligament, containing the left gastroepiploic . . . the vasa brevia, . . . bursa is opened and the splenic vessels are seen running along the superior margin of the tail of the pancreas to the hilus of the spleen (Fig. 515).

The splenic artery is usually isolated for 1 cm. or so at a convenient site and a tie of 0 silk is placed about it in continuity so that during the rest of the operation splenic blood will be returning to the general circulation (Fig. 515). The spleen will decrease in size quite remarkably.

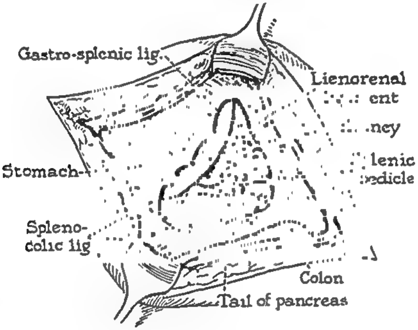

Fig. 519.—Splenectomy. The completed operation.

A hand is passed over and behind the spleen peeling off the attachments to the posterior parietal peritoneum and diaphragm. The thicker ones are clamped and tied. The spleen is lifted into the wound and a saline pack tucked behind it into the fossa to control oozing and to prevent the spleen from falling back. The lienorenal and splenocolic ligaments are exposed behind and below the spleen and are divided (Fig. 516). The spleen is swung outward and downward, and the remaining gastrosplenic ligament clamped and divided (the collateral circulation here may be tremendous when the spleen is large) (Fig. 517). Only the pedicle remains intact. It is separated in front and behind from the tail of the pancreas. The artery and vein are isolated, separately clamped and divided, and doubly tied with suture-ligatures of 0 silk (Fig. 518). If the splenic artery was not clamped in the beginning of the operation, the artery is clamped first at this point and the spleen is massaged gently to empty it of blood before the vein is clamped.

Before the abdomen is closed a search is made for accessory spleen or spleens, inspecting especially the retroperitoneum about the pedicle stump and pancreas, the splenocolic ligament, the greater omentum and the intestinal mesentery.

CHAPTER 63

DUODENAL OBSTRUCTION

By A. V. Partipilo*

HISTORICAL

The first mention of the subject was made by Calder of Edinburgh, in 1732, who described a case of congenital obstruction of the duodenum. The first authentic report of a case of duodenal obstruction in the adult is that of Boernerus in 1752. This was written in Latin and translated into the English language by J. W. Brannan. This case is of sufficient interest to warrant a brief resume. The patient was a man, over fifty years of age, whose chief complaint was nausea, vomiting, and constipation. The patient was extremely emaciated, and in about eight weeks he died. Boernerus made a post-mortem examination and found, much to his surprise, a greatly distended stomach filling the abdomen from the diaphragm to the pubes. Upon further investigation he found that over the duodenum there appeared a solid and wrinkled constriction, exceeding the thumb in breath. This cord was so contracted that he was not able to inject through the duodenum even "the most dilute fluids." Boernerus concluded as follows: "Behold then a most rare but clear case (the truth of which, were I not an eye-witness, I should myself doubt). Behold a dry fatal tabes, caused by a remarkable stricture of the duodenum with an abnormal position of the stomach."

Yeats, in 1820, made some observations on the connections of the duodenum and its relationship to the liver, and illustrated his findings with descriptive plates. He described toxic symptoms which he attributed to compression of the duodenum by the transverse colon. Guyot, in 1829, reported a case of congenital contraction of the duodenum, and 1833, Duplay, a case of acute dilatation of the stomach.

The first American publication on the subject was contained in an article by J. C. Anderson, written in 1848, entitled "Stricture of the Duodenum." Miller and Humby, in 1853, published the first case in the English language of "an enormously dilated stomach which after death was found to contain five basinsful of fluid." According to Novack, Brintonnin 1859, was the first to suggest that gastric dilatation was due to a reflex nervous mechanism.

As early as 1842, Rokitansky recognized the possibility of acute gastro-mesenteric obstruction. In the third edition of his book, which appeared in 1861, he described the mechanism of acute dilation of the stomach as being due to compression of the duodenum by the mesenteric vessels. This view was elaborated and modified by a host of others. Heschl, 1851, described the mechanism in more detail; Bamberger, 1855, associated the condition with acute infections; Erdman, 1868, *associated gastric dilatation as a result of a fall or injuries;* Fagge, 1873, described the symptoms of acute dilatation of the stomach; Morris, 1873, reported a case of acute gastric dilatation which he thought was due to excessive secretion of

* The material for this chapter is taken mainly from an article "Duodenal Obstruction" by A. V. Partipilo and G. A. Wiltrakis, "Surgery," April 1942.

the stomach; and Glenard, 1889, pointed out the possibility of gastric dilatation being due to narrowing of the duodenojejunal angle by the sinking of the small intestine into the true pelvis.

Credit for the modern conception of the pathogenesis of arteriomesenteric obstruction belongs to Albrecht, who in 1889 reported two cases of chronic duodenal compression. He observed a flattening of the duodenum between the mesenteric pedicle and the spine. By attaching weights to the mesentery he demonstrated that a downward and backward traction constricted the duodenum, and found that a considerable amount of pressure was required to force water beyond the constriction.

Petit, in 1900, made some anatomical studies and found that a groove was normally present on the terminal duodenum which was made by the mesenteric pedicle. He presented two drawings to support his theory that duodenal obstruction was due to traction on the mesentery, and that the condition was an exaggeration of a normal relationship. He was the first to suggest a duodenojejunostomy to correct the condition. Robinson, in 1900, wrote an extensive paper on the subject and reported some cases which he observed during the course of autopsies. He operated upon two patients which revealed no other lesion than a dilatation of the duodenum. Mueller, 1900, was of the opinion that the horizontal position of the patient, and preoperative purgation caused the small intestine to collapse and sink into the pelvis, thus predisposing to acute gastric dilatation. Finney, 1902–1905, was not certain but that gastro-mesenteric ileus and acute gastric dilatation might be one and the same thing. He pointed out that the characteristic feature of each was dilatation of the stomach, and the findings at autopsies were quite constant; namely dilatation of the stomach and duodenum up to the point where it is crossed by the superior mesenteric vessels. Zade, 1905, in a discussion of postoperative ileus stated that dilatation of the stomach was the most important factor in the pathogenesis of duodenal compression. This latter view was supported by Dragstedt, et al, who held the view that gastric dilatation is due primarily to reflex inhibition of the peripheral gastric motor mechanism, and that duodenal compression is secondary to a direct pressure of the dilated stomach.

Morris, in 1905, called attention to adhesions in the region of the gall bladder, which he called "cobwebs in the attic of the abdomen," as factors in duodenal obstruction. In 1914, M. L. Harris reviewed the subject and reported [illegible] cases of duodenal obstruction due to bands which bridged across the duodenum from the gall bladder to the transverse colon. Lane wrote extensively on the subject and stressed that adhesions frequently were predisposing factors to duodenojejunal obstruction. Duval, 1922, gave the name of "essential periduodinitis" in those cases in which the actual cause for the production of adhesions was not found, but the adhesions in themselves provoked incomplete compression of the duodenum.

Conner in 1906 [illegible] ing Albretch's observatio[illegible] : to the mesenteric pedicle and then passed it through the anus. By applying weights to the end of the cord he found it was necessary to add 500 grams, which was equivalent to the approximate weight of the intestine, to constrict the duodenum and prevent passage of water under pressure of 20 to 48 mm. of mercury. Conner's article deals extensively with acute duodenal obstruction and its perusal is worth while.

In America, duodenojejunostomy was first suggested by Barker in November, 1905, at a meeting of the Johns Hopkins Society while discussing Finney's paper.

Bloodgood also suggested the operation in 1906; however, Stavely, in 1907, is accredited with having performed the first duodenojejunostomy in the adult, while Ernst, in 1916, reported the first operative recovery following this operation in an eleven-day old infant.

Codman, in 1908, in a very extensive review of the subject compared the arteriomesenteric crotch of the quadruped posture and the erect posture in man. He stated that the arterial crotch in man became closed when the body was in the erect because of the vertical position assumed by the mesentery, and that anatomical or physiological variations from the normal may increase the pressure to the extent of causing a compression of the duodenum (Fig. 520). He believed that this ab-

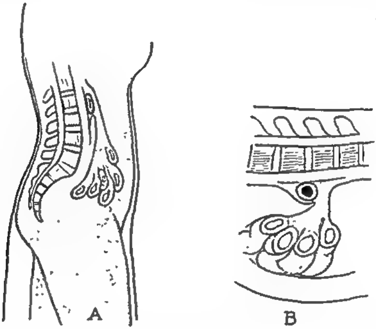

Fig. 520.—Comparison of the arteriomesenteric crotch in the quadruped (b) posture and the erect posture in man.

normal relationship may have a bearing on the etiology of peptic ulcer and gall bladder disease. In this latter connection, Sloan in 1923, reported 54 cases of peptic ulcer which at operation revealed a partial obstruction at or near the duodenojejunal angle. Surgical relief of the obstruction was sufficient in most cases to cure the ulcer.

Bloodgood, in 1907, associated a large prolapsed cecum and a short mesentery of the ileum with duodenal obstruction, and in 1912 he resected the right half of the colon for this condition. As an alternative procedure, colopexy was suggested by Wilkie in 1922.

In recent times a considerable amount of literature has accumulated dealing with various phases of duodenal obstruction. In 1917, Vanderhoof reviewed the literature and reported 6 cases, and in 1923 reported 22 additional cases of visceroptosis as the cause of chronic duodenal obstruction. E. L. Kellogg has been the most prolific contributor on the subject. His first article appeared in 1918, and since then he has written numerous articles including a textbook (1933) which deals extensively with every phase of duodenal diseases. Partipilo and Wiltrakis, in 1942, classified the disease and described the pathogenesis in the production of duodenal obstruction.

A considerable number of articles have been devoted to duodenal obstruction in the newborn. The articles of Webb and Wangensteen, Ladd, and Stenson cover the bibliography rather thoroughly. Stenson's article, 1938, revealed that out of the several hundred cases reported by various authors there were only 15 operative recoveries. He stated that in several hundred instances when an enterostomy was done, the termination was fatal. He concluded that the safest procedures, when indicated, are: gastrojejunostomy, duodenojejunostomy, and iliocolostomy. Ladd, in 1932, reported ten cases of congenital duodenal obstruction and made an exhaustive review of the literature, and in 1937, he described the symptoms, signs, X-ray findings and treatment.

DEFINITIONS

Duodenal obstruction, in its broadest sense, may be defined as an impediment or hindrance to the forward passage of the duodenal contents caused by acquired or congenital conditions. The obstruction may be acute or chronic, complete or incomplete, the characteristic feature being a dilated duodenum.

A survey of the literature reveals a lamentable lack of unanimity in respect to the nomenclature of duodenal obstruction. The following are some of the multitudinous terms which have been applied to describe the disease; intermittent duodenal obstruction, duodenal ileus, mesenteric ileus, gastromesenteric ileus, duodenal stasis, stenosis of the duodenum, megaduodenum, arteriomesenteric occlusion, dilatation and stasis of the duodenum, chronic compression of the duodenum, etc. It is obvious that these terms may be appropriately used to describe specific or individual types of obstruction, however when used as all-inclusive titles the result is confusion, and misunderstanding.

Frequently, this syndrome is referred to as an ileus. The word ileus is derived from the Latin, meaning to twist. At the present time, as a result of common usage, ileus is applied to designate an obstruction due to paralytic conditions of the bowel in contradistinction to that due to mechanical causes. To avoid confusion, the term ileus should be restricted to obstruction due to a disturbance in the nervous mechanism.

Mesenteric obstruction (or ileus) is a term which is most commonly applied loosely, inaccurately, and without forethought. Possibly, the reason for this is due to the fact that traction by the mesenteric pedicle is very often the cause of the obstruction. However, other factors may be the cause, such as embryonal bands, congenital malformations, extrinsic and intrinsic tumors of the duodenum, etc. Hence, the term mesenteric obstruction does not appropriately describe the condition in all cases.

Gastromesenteric ileus, acute dilatation of the stomach, and duodenal obstruction are often used interchangeably and synonymously. This is erroneous because the three conditions may occur independently. Experience has indicated to us that gastric dilatation associated with duodenal obstruction was conspicuous by its absence. We believe that gastric dilatation and duodenal obstruction are two distinct clinical entities and for the sake of clarity the two conditions should be dissociated from each other. The term "duodenal obstruction" is self-explanatory, therefore readily understood. It is all inclusive and describes the syndrome in every case regardless of the etiological factor or its pathogenesis.

ETIOLOGICAL CLASSIFICATION

Duodenal obstruction may occur at any age, afflicting the male and female alike, possibly the female more often. Almost every possible factor in the causation of the disease has been reported. These factors may be classified into two general groups: (1) those which are basically of congenital origin; and (2) those which are acquired. It is obvious that the two groups overlap in many instances. Very often the predisposing factor is acquired, whereas, the basic cause in the pathogenesis of the obstruction is of congenital origin. For instance, arteriomesenteric obstruction is in reality an accentuation of a normal condition, hence basically congenital, while the predisposing factors are usually acquired. This type of obstruction should be placed in the congenital group with the reservation that it is not truly of congenital origin. Bearing these equivocations in mind, the following classification is given.

I. Congenital Duodenal Obstruction
  - A. Arteriomesenteric (compression of duodenum by the mesenteric pedicle). This type is influenced by the following predisposing factors:
    1. Ptosis of small intestine due to loss of mesenteric fat.
    2. Abdominal relaxation following pregnancy and debilitating diseases.
    3. Ptosis of the large bowel.
    4. Lordosis of the lumbar spine.
  - B. Mesocolic. In this variety of obstruction the duodenum becomes compressed by the mesocolic root alone. The condition is very often associated with prolapsed hepatic flexure of the colon.
  - C. Arteriomesocolic. In this type the duodenum is compressed by the mesocolic root and the middle colic vessels. It is associated with a mobile cecum and a prolapsed ascending and hepatic flexure.
  - D. Extrinsic duodenal atresia (complete gap in duodenal continuity), the partition may be due to:
    1. Arrested development.
    2. Vascular changes.
    3. Inflammatory effects.
  - E. Extrinsic duodenal occlusion.
    1. Anomalous mesenteric vessels occluding the duodenum (Buchanan).
    2. Anomalous annular pancreas.
    3. Abnormal embryonal bands.
       - a. Hepaticoduodenal (Fig. 522 *A*).
       - b. Hepaticoduodenocolic (Fig. 522 *E*).
       - c. Duodenoduodenal (agglutination of the first upper half to the second portion of the duodenum). (See Fig. 522 *D*.)
       - d. Duodenorenal.
       - e. Jejunomesocolic (Mayo's band) and lateral attachment of the first loop of jejunum to the transverse mesocolon reported by Partipilo and Wiltrakis.
    4. Contraction of ligament of Treitz, drawing the duodenaljejunal angle upward.
    5. Faulty rotation of duodenum may produce complete or incomplete occlusion.

6. Intraperitoneal mobile duodenum.
   a. With omentum and ascending colon attached to duodenum and jejunum (Bargen and Walters).
   b. With complete interruption of duodenum (Kellogg).

II. Acquired Duodenal Obstruction.
   A. Inflammatory adhesions.
      1. Due to infection of gall bladder.
      2. Essential periduodinitis of Duval.
      3. Hepaticoduodenocolic bands as a result of gall bladder, colonic, or gastric disease.
      4. Fixation by adhesions of the transverse colon into the true pelvis associated with sinking of the small intestine (Partipilo and Wiltrakis).
      5. Contracting adhesions from peptic ulcers or pancreatitis.
   B. Extrinsic compression.
      1. Enlarged inflammatory lymph glands.
      2. Lymphatic enlargement from Hodgkin's disease and invasion of the duodenum (Partipilo and Wiltrakis).
      3. Aneurysm of the abdominal aorta (Partipilo and Wiltrakis).
      4. Aneurysm of the superior mesenteric artery (Partipilo).
      5. Kinking and angulation of the duodenum following gastric operations.
      6. Secondary compression of the duodenum by a dilated stomach (Dragstedt).
   C. Intrinsic occlusion.
      1. Gall bladder stones and foreign bodies.
      2. Carcinoma of the duodenum.
      3. Intussusception due to pyloric or duodenal polyp (Kellogg).
   D. Extraduodenal.
      1. Jejunal dilatation due to compression at the pelvic brim when small intestine is tightly wedged into the pelvis (Kellogg).
      2. Jejunomesocolic inflammatory bands attaching the jejunum to mesocolon (Partipilo).

PATHOLOGIC ANATOMY

The duodenum is from 10 to 12 inches long and extends from the pylorus to the duodenojejunal flexure. It varies from a U to a C-shape, with the concavity of its curvature directed upward and to the left. The duodenum may be divided into four parts: (1) superior horizontal; (2) descending; (3) inferior horizontal; and (4) ascending or terminal.

The first part measures about 2 inches in length and is a direct continuation of the pylorus. It is completely surrounded by peritoneum and has some mobility. Its course is backward, to the right, and slightly upward, terminating downward into the descending portion. It forms the lower border of the foramen of Winslow and is in contact with the under surface of the left lobe of the liver and the neck of the gall bladder.

The descending portion is from 3 to 4 inches long. It descends along the vertebral bodies to the level of the middle third lumbar vertebra, where it turns

to the left and becomes continuous with the third part. It is covered by peritoneum on its anterior surface except at its middle portion where it is crossed by the root of the mesocolon. Figure 521 is a schematic illustration showing the relationships of the duodenum. Note that the root of the mesocolon is shown as dividing the duodenum into an upper, supramesocolic, and a lower, inframesocolic part. This

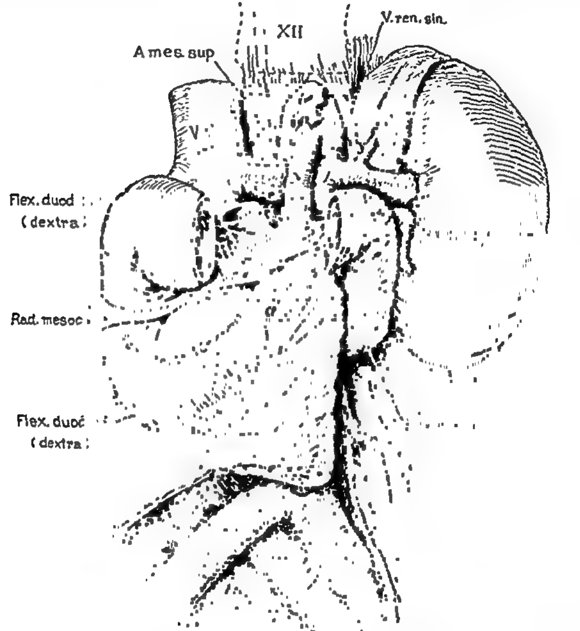

FIG. 521.—Drawing showing the topographical relationship of the duodenum, the left renal vein, and the superior mesenteric artery. (Courtesy of Bela Halpert).

relationship is of clinical interest as the mesocolic root may be a factor in compressing the duodenum at this level. This is not generally appreciated nor generally understood. Bloodgood (1912) was of the opinion that this type of obstruction was due to traction on the mesenteric pedicle as a result of a dilated cecum displaced into the pelvis, and that tension on the mesentery of the small bowel could not occur unless the last portion of the ileum had an unusually short mesentery. Ac-

cording to Halpert (1926), "the last portion of the ileum always has a very short mesentery, or better, it never has one of any considerable length unless the cecum is movable. The occlusion of this type is caused by the mesocolon and not by the mesentarium, and is therefore rather an 'arteriomesocolic' than an arteriomesenteric occlusion." He further stated that, "traction on the superior mesenteric artery is also present and is exerted by the ileocolic, right colic, and middle colic arteries and not by the jejunal arteries as occurs in arteriomesenteric obstruction." In conformity with the desire to clarify the nomenclature, the term mesocolic ob-

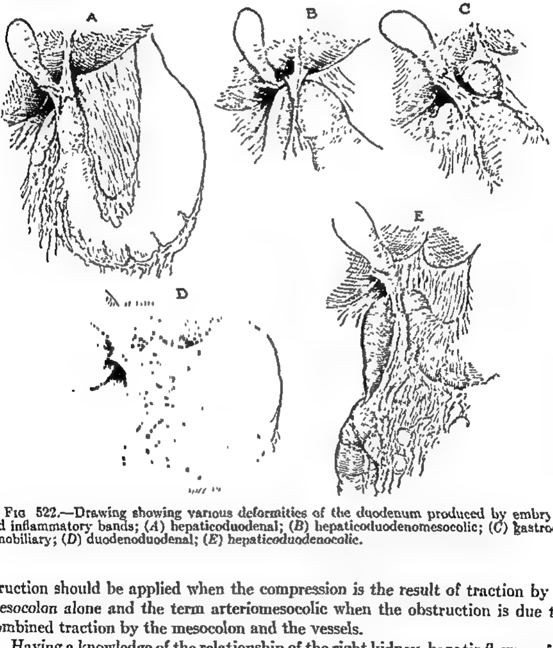

FIG 522.—Drawing showing various deformities of the duodenum produced by embryonal and inflammatory bands; (*A*) hepaticoduodenal; (*B*) hepaticoduodenomesocolic; (*C*) gastroduodenobiliary; (*D*) duodenoduodenal; (*E*) hepaticoduodenocolic.

struction should be applied when the compression is the result of traction by the mesocolon alone and the term arteriomesocolic when the obstruction is due to a combined traction by the mesocolon and the vessels.

Having a knowledge of the relationship of the right kidney, hepatic flexure of the colon, and the gall bladder to the first and second portions of the duodenum makes it easy to understand how malformations or ptosis of the kidney and colon and bands of adhesions may drag upon and distort the duodenum to produce varying degrees of obstruction. Figure 522 illustrates various deformities of the duodenum that may result from embryonal or inflammatory bands.

The inferior horizontal part of the duodenum crosses the abdominal wall at the level of the third lumbar vertebra, from the right to the left. Its direction is

and the drag upon the mesenteric pedicle by the vessels supplying the right half of the colon. Figure 524 illustrates duodenal dilation resulting from traction by the middle colic artery, following ptosis of the right half of the colon and hepatic flexure. The author reported a case of duodenal obstruction due to drag on the middle colic artery as a result of a prolapsed transverse colon. In this case (Fig. 529) the transverse colon was firmly adherent by adhesions to the true pelvis, dragging the middle colic artery vertically downward and backward in the same plane as the mesenteric pedicle of the small intestine. The duodenum was found greatly distended, and we believe that its compression was due to traction by the middle colic artery, and not by the pedicle.

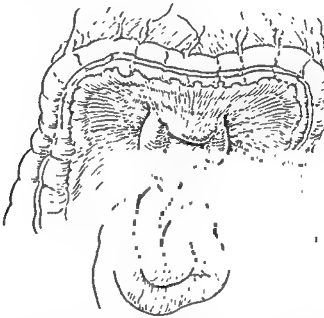

FIG. 525.—About 3 inches of the jejunum is attached to the transverse mesocolon by an embryonal peritoneal band (author's case, "Surgery," April, 1912).

There is considerable controversy concerning the pathogenesis of acute gastric dilatation. Two theories have been proposed: (1) that the dilatation is primarily a reflex nervous disturbance; and (2) that dilatation of the stomach is associated with, or is secondary to, arteriomesenteric obstruction. Kellogg (1935) recognized the possibility of acute gastric dilatation, but believed that in the majority of instances mesenteric obstruction was also found. He thought that the essential pathology was compression of the third part of the duodenum by the mesenteric pedicle. He stated that gastric dilatation may also be produced by kinks at the superior and duodenal-jejunal angles, and occasionally by compression of the jejunum at the pelvic brim when the small intestines are tightly wedged into the pelvis. On the other hand, Dragstedt and his co-workers concluded that gastric dilatation following operation was due to a reflex inhibition of the peripheral gastric motor mechanism through impulses reaching the stomach by way of the vagi and splanchnics. They believed that an enlarged duodenum, if present, was secondary, and was caused either by a direct pressure of the dilated stomach on the transverse duodenum, or by a secondary arteriomesenteric compression brought about by the downward pressure of the dilated stomach which forced the intestine into the pelvis (Figure 526.)

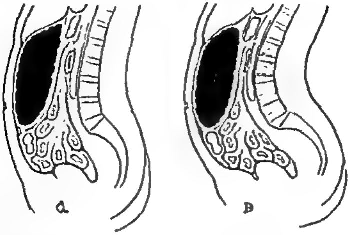

FIG. 526.—(a) Illustrates how dilatation of the stomach forces the small intestine into the pelvis, producing a downward and backward traction on the mesenteric pedicle; and (b) secondary duodenal compression (after Dragstedt, et al.).

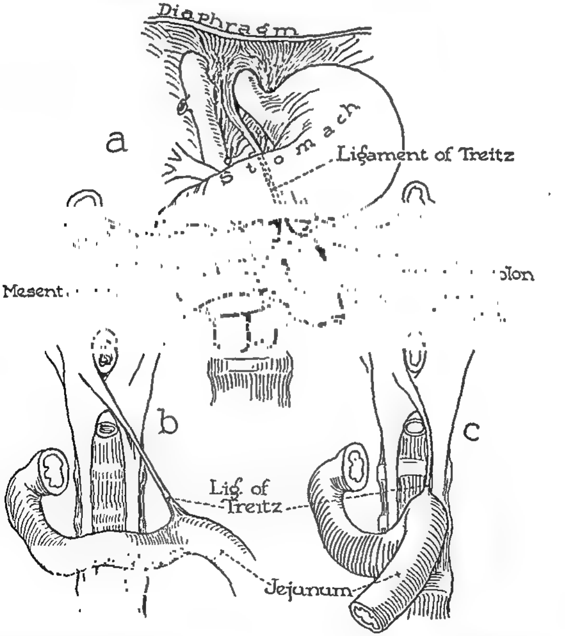

FIG. 527—Schematic representation of the duodenum and its relationship. (b) Elongated ligament of Treitz (c) Short ligament which draws the duodenojejunal flexure rather high and to the left, thus rendering the flexure more acute

Duodenal obstruction not infrequently occurs as a result of acute angulation of the duodenojejunal flexure. Its pathogenesis is based upon a disturbance of the normal relationship of the duodenum and the ligament of Treitz. When the terminal duodenum reaches the left side of the first or second lumbar vertebra, it turns abruptly forward, downward, to the left, and passes into the jejunum. This bend, known as the duodenojejunal flexure, is fixed by a thin band of unstriped muscle called the ligament of Treitz. The latter is attached to the left crus of the diaphragm, and is continuous with the duodenal musculature. Its relative length determines the position of the duodenojejunal flexure. If short, the flexure is placed rather high and to the left, thus rendering the flexure more acute (Fig. 527c).

FIG. 528.—Mayo's band. The first portion of the jejunum is fixed to the inferior surface of the mesocolon, thus acutely angulating the duodenojejunal flexure by pulling the jejunum to the right of the body. (Partipilo, "Surgery," April, 1942).

The acuteness of the angulation may be accentuated by a gastrojejunostomy, especially when the patient is in Fowler's or semi-sitting position. For this reason the surgeon should examine the duodenojejunal flexure in all cases when a posterior gastrojejunostomy is contemplated. If found acutely angulated, the ligament should be divided and its end ligated.

Duodenal obstruction may also result from an abnormal attachment of the first loop of jejunum. In 1908 Mayo described the attachment of the first loop of jejunum to the transverse mesocolon by bands of adhesions. As illustrated in Figure 528, this band unites the inferior surface of the mesocolon to the anterior surface (right side) of the jejunum, thus pulling the jejunum to the right and acutely angulating the duodenojejunal angle. A very unusual abnormality of the first loop of jejunum was reported by the author. As illustrated in Figure 525, about three inches of the jejunum was firmly attached to the transverse mesocolon by a peritoneal band.

The literature is replete with reports of unusual cases of duodenal obstruction. Henske and Best in 1928, reported a case of congenital duodenal obstruction in an infant six months old which was due to incomplete rotation of the intestine on its

mesenteric axis. E. L. Kellogg, in 1931, reported an unusual case of *intussusception* of the duodenum in a man sixty-nine years old, caused by a pedunculated adenoma originating in Brunner's *glands of the duodenum*. The tumor, attached to the posterior duodenal wall *close to the pyloric ring*, had migrated down to the upper portion of the jejunum causing intussusception of the duodenum and pyloric end of the stomach. Anomalous pancreas as the cause of obstruction was probably first described by Ecker in 1862. Bargen and Walters, in 1933, operated on a fourteen year old girl whose duodenum instead of being retroperitoneal was intraperitoneal. The duodenojejunal angle was directed to the outer side of the ascending colon, and as a result the omentum *and ascending mesocolon* were adherent to the jejunum and duodenum. All portions of the duodenum, particularly the second part, assumed a size equal to a dilated large intestine. These and many other cases indicate the unusual possibilities that may be present to cause duodenal obstruction.

DIAGNOSIS

Although this text is not concerned with clinical features of disease, nevertheless a short resume of the findings is given because surgical textbooks are painfully silent on the subject of duodenal obstruction. This is due to the assumption that the duodenum *is surgically the silent portion of the gastrointestinal tract. This is* contrary to our experience.

The diagnosis of duodenal obstruction may be easily arrived at in some cases, while in others it may not be so obvious. No single symptom or combination of symptoms and physical findings is pathognomic. However, when these symptoms and findings are analyzed and correlated with the history of the case, and supplemented with roentgen studies, they assume definite characteristics indicative of duodenal obstruction.

The history of chronic duodenal obstruction reveals that the age-group is in *the third decade, and that it occurs more often in the female than in the male* (the ratio is 3 to 1). These patients are the visceroptotic type; asthenic, undernourished, thin individuals with scaphoid abdomens. A great majority of these patients have had previous medical treatment for "stomach trouble," peptic ulcer, gall bladder disease, chronic appendicitis, "dyspepsia," "nervous stomach," "vagatonia," neurasthenia, or other chronic abdominal ailments. Many will give a history of having had one or more laparotomies, which not only failed to relieve them but very often aggravated the *original symptoms. The physician should be on the alert and* investigate the duodenum when a patient gives a history of vague upper abdominal symptoms, or who has been treated for gastric neurosis, vagatonia, etc. It is our belief that in some cases the underlying cause of neurasthenia is an obscure disturbance of one of the body systems, such as the reproductive, endocrine, genitourinary, gastric, etc. *If a "neurasthenic" patient presents a history predominated* by gastric symptoms, it is logical to rule out duodenal pathology.

Patients with chronic obstruction involving the first and second portion of the duodenum may be well developed and not have the physical characteristics of the asthenic type as seen in the arteriomesenteric obstruction. The symptoms and physical findings *in chronic obstruction* of the first and second portion of the duodenum are similar to those accompanying duodenal ulcer or gall bladder disease. Therefore, these are often wrongly diagnosed as peptic ulcer or chronic cholecystitis. In spite of the similarity of the symptoms, two important facts stand out: (1)

medical treatment for peptic ulcer or for the gall bladder disease fails to relieve the symptoms, and (2) roentgen studies invariably reveal a duodenal deformity.

A host of symptoms descriptive of duodenal obstruction have been enumerated by various writers. The following is a more or less comprehensive list: vomiting, nausea, bilious attacks, migraine headache, constipation, loss of weight, eructation, epigastric pain, regurgitation, borborygmus, dragging sensation, etc. One can readily observe that these symptoms taken singly or collectively are suggestive of any number of abdominal diseases, and not conclusive of any. However, when a combination of these symptoms are analyzed in relation to the duration of the illness, onset and course of the symptoms, and correlated with physical and x-ray findings, they assume certain features characteristic of duodenal obstruction, and become distinguishable from other abdominal conditions.

FIG 529.—Compression of the duodenum by the middle colic artery. Five-hour plate shows 60 per cent residue in the stomach with barium in the duodenum and duodenal cap. (Partipilo, "Surgery," April, 1942)

The history reveals that in chronic duodenal obstruction the illness is of long duration. Many patients will date back their complaints since childhood. Another characteristic feature is the intermittent recurrent nature of the attacks. In some individuals these attacks may be in the nature of "gastric disturbances," such as constipation, nausea, headache, and "bilious attacks," while in others they may be ushered in with nausea, vomiting, epigastric pain and other symptoms indicative of high intestinal obstruction. Usually the attacks may not be particularly severe until early adult life when a debilitating disease is followed by loss of mesenteric fat or frequent pregnancies by abdominal relaxation.

Of the symptoms enumerated, vomiting is the most characteristic. It is generally intermittent and the vomitus contains enormous quantities of bile. When the obstruction is complete, the emesis is abundant, continuous, and occurs soon

after a meal is eaten. The attack may last for a few hours or for days depending upon the degree of the compression. At times, vomiting is relieved by assuming the knee-chest position, or by lying on the abdomen or on the right side. This is a significant factor to elicit in the history since it is only in duodenal obstruction that posture relieves the patient of vomiting. This may be used as a therapeutic test. By having the patient rest in bed with the foot of the bed elevated 18 inches usually brings relief.

Epigastric pain is associated with vomiting. It varies from a mild discomfort to a sharp colicky pain. The patient is relieved or the pain subsides after vomiting. The pain in duodenal obstruction is probably due to the distended duodenum and to the unyielding spastic pylorus. When the pylorus yields, the duodenal contents empty into the stomach and the patient vomits. Thus relief of pain which fol-

FIG. 530.—Compression of the [illegible]

lows is possibly the result of pyloric relaxation and relief of intraduodenal pressure. The process is a vicious circle as the pain will recur with subsequent accumulation of secretion in the duodenum. It should be stressed that acute duodenal obstruction ushered in without a previous history of gastric disturbances is usually due to occlusion of the duodenum by intrinsic or extrinsic tumors.

X-RAY EXAMINATION

Careful roentgen studies will confirm the diagnosis in practically every case. It should be emphasized, however, that if these studies are made in between the attacks; when the patient is free from symptoms, the roentgen observations may be negative. Roentgen observations were first reported by Jordan in 1911, who described, and showed x-ray plates, the findings of duodenal obstruction. These

findings were verified by Lane. Jewett, quoted by Lichty, insisted that certain essential points are necessary to establish the diagnosis. He said: "The essential points then are dilatation of the duodenum, writhing of the duodenum, waves or reverse peristalsis, and puddling at the duodenal jejunal flexure. There is usually a low descending loop in the duodenum and a high point of fixation of the duodeno-jejunal angle which can be demonstrated by lifting of the stomach. There is often a six-hour gastric stasis, and the duodenal cap is not infrequently dilated along with the rest of the duodenum. The diagnosis, however, is made largely on the fluoroscopic findings."

Kellogg and Kellogg, in 1927, noted that changes in the duodenum follow one or four types:

1. Asthenic duodenum (symptoms latent or toxic). Roentgen examination may show delay or puddling in the duodenum, with sluggish peristalsis and slight or no dilatation

Fig. 531

Fig. 532

Fig. 531.—Carcinoma of the terminal duodenum. Five-hour plate shows retention in the duodenum. (Partipilo, "Surgery," April, 1942).

Fig. 532.—Lymph glands (Hodgkin's disease) invading and obstructing the terminal duodenum. Five-hour plate shows an enormously enlarged duodenum. Arrow indicates the sharply demarcated duodenum. (Partipilo, "Surgery," April, 1942).

2. Duodenal obstruction, with incompetent pylorus. Bile regurgitates easily into the stomach. Dilatation is usually moderate or absent. Roentgen examination is negative or shows reverse peristalsis.

3. Obstruction with hypertrophy (the writhing duodenum). The duodenum is elongated and its walls are thickened. Under the fluoroscope it is seen to labor over its contents.

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Shattuck and Imboden have made the following observations:

1. Bands about the first and second portion of the duodenum may show the duodenum markedly contracted in size in comparison with the size of the stomach, or it may be larger than normal. The duodenum may be deformed, as in ulcer, but the deformity is not constant and varies from time to time in various positions.

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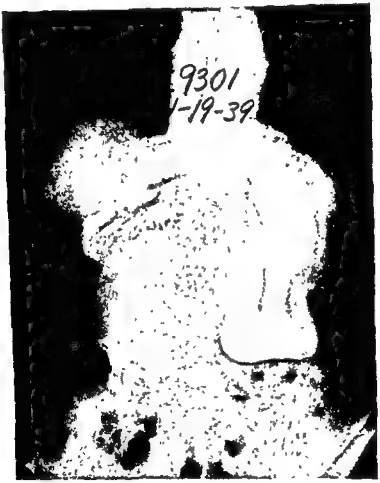

FIG. 533.—Aneurism of the abdominal aorta compressing the terminal duodenum. Immediate plate showing the duodenum filled with barium up to the spine. Note the displacement of the stomach caused by the pressure of the aneurism. (Partipilo, "Surgery," April, 1942).

3. A commonly observed phenomena is a fixation of the dependent limb of the duodenum to the liver at a certain point, producing a definite angulation.

4. Obstruction at the ligament of Treitz shows the angle is shifted to the right of the normal position. The dependent duodenum is usually enlarged, especially in its transverse diameter, and the valvulae conniventes are frequently absent.

To summarize, the findings and technique of fluoroscopic examination are: The examination is made while the patient is standing. After the stomach has been examined, it is pushed upward and to the left by the examiner's hand, thus gaining an unobstructed view of the duodenum. Barium will be seen to enter the duodenum rather rapidly at first; however, as soon as it reaches the lumbar vertebrae, it stops. The duodenum becomes filled and is seen to enlarge, soon the barium from the

after a meal is eaten. The attack may last for a few hours or for days depending upon the degree of the compression. At times, vomiting is relieved by assuming the knee-chest position, or by lying on the abdomen or on the right side. This is a significant factor to elicit in the history since it is only in duodenal obstruction that posture relieves the patient of vomiting. This may be used as a therapeutic test. By having the patient rest in bed with the foot of the bed elevated 18 inches usually brings relief.

Epigastric pain is associated with vomiting. It varies from a mild discomfort to a sharp colicky pain. The patient is relieved or the pain subsides after vomiting. The pain in duodenal obstruction is probably due to the distended duodenum and to the unyielding spastic pylorus. When the pylorus yields, the duodenal contents empty into the stomach and the patient vomits. Thus relief of pain which fol-

FIG. 530.—Compression of the duodenum by the middle colic artery. Postoperative plate made on the 40th postoperative day. Note the barium passing directly into the jejunum through—the duodenojejunostomy, (Partipilo, "Surgery," April, 1942).

lows is possibly the result of pyloric relaxation and relief of intraduodenal pressure. The process is a vicious circle as the pain will recur with subsequent accumulation of secretion in the duodenum. It should be stressed that acute duodenal obstruction ushered in without a previous history of gastric disturbances is usually due to occlusion of the duodenum by intrinsic or extrinsic tumors.

X-RAY EXAMINATION

Careful roentgen studies will confirm the diagnosis in practically every case. It should be emphasized, however, that if these studies are made in between the attacks; when the patient is free from symptoms, the roentgen observations may be negative. Roentgen observations were first reported by Jordan in 1911, who described, and showed x-ray plates, the findings of duodenal obstruction. These

medical treatment for peptic ulcer or for the gall bladder disease fails to relieve the symptoms, and (2) roentgen studies invariably reveal a duodenal deformity.

A host of symptoms descriptive of duodenal obstruction have been enumerated by various writers. The following is a more or less comprehensive list: vomiting, nausea, bilious attacks, migraine headache, constipation, loss of weight, eructation, epigastric pain, regurgitation, borborygmus, dragging sensation, etc. One can readily observe that these symptoms taken singly or collectively are suggestive of any number of abdominal diseases, and not conclusive of any. However, when a combination of these symptoms are analyzed in relation to the duration of the illness, onset and course of the symptoms, and correlated with physical and x-ray findings, they assume certain features characteristic of duodenal obstruction, and become distinguishable from other abdominal conditions.

[illegible] middle colic artery. Five-hour plate shows the duodenum and duodenal cap, (Partipilo, [illegible]

The history reveals that in chronic duodenal obstruction the illness is of long duration. Many patients will date back their complaints since childhood. Another characteristic feature is the intermittent recurrent nature of the attacks. In some individuals these attacks may be in the nature of "gastric disturbances," such as constipation, nausea, headache, and "bilious attacks," while in others they may be ushered in with nausea, vomiting, epigastric pain and other symptoms indicative of high intestinal obstruction. Usually the attacks may not be particularly severe until early adult life when a debilitating disease is followed by loss of mesenteric fat or frequent pregnancies by abdominal relaxation.

Of the symptoms enumerated, vomiting is the most characteristic. It is generally intermittent and the vomitus contains enormous quantities of bile. When the obstruction is complete, the emesis is abundant, continuous, and occurs soon

after a meal is eaten. The attack may last for a few hours or for days depending upon the degree of the compression. At times, vomiting is relieved by assuming the knee-chest position, or by lying on the abdomen or on the right side. This is a significant factor to elicit in the history since it is only in duodenal obstruction that posture relieves the patient of vomiting. This may be used as a therapeutic test. By having the patient rest in bed with the foot of the bed elevated 18 inches usually brings relief.

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findings were verified by Lane. Jewett, quoted by Lichty, insisted that certain essential points are necessary to establish the diagnosis. He said: "The essential points then are dilatation of the duodenum, writhing of the duodenum, waves or reverse peristalsis, and puddling at the duodenal jejunal flexure. There is usually a low descending loop in the duodenum and a high point of fixation of the duodeno-jejunal angle which can be demonstrated by lifting of the stomach. There is often a six-hour *gastric stasis, and the duodenal cap is not infrequently dilated along* with the rest of the duodenum. The diagnosis, however, is made largely on the fluoroscopic findings."

Kellogg and Kellogg, in 1927, noted that changes in the duodenum follow one or four types:

1. Asthenic duodenum (symptoms latent or toxic). Roentgen examination may show delay or puddling in the duodenum, with sluggish peristalsis and slight or no dilatation.

Fig. 531

Fig. 532

Fig. 531.—Carcinoma of the terminal duodenum. Five-hour plate shows retention in the duodenum, (Partipilo, "Surgery," April, 1942).

Fig 532.—Lymph glands (Hodgkin's disease) invading and obstructing the terminal duodenum. Five-hour plate shows an enormously enlarged duodenum. Arrow indicates the sharply demarcated duodenum, (Partipilo, "Surgery," April, 1942).

2. Duodenal obstruction, with incompetent pylorus. Bile regurgitates easily into the stomach. Dilatation is usually moderate or absent. Roentgen examination is negative or shows reverse peristalsis.

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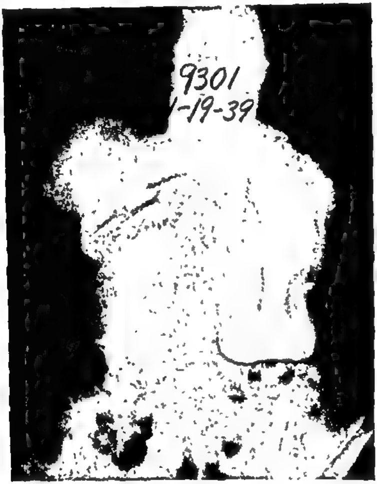

FIG. 533.—[illegible] liate plate showing [illegible] the stomach cause[illegible]

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stomach stops flowing into the duodenum, and instead, as a result of an active antiperistalsis, there is regurgitation of the duodenal contents into the stomach. The duodenum may appear to be contorted or writhing, and the contents may be seen to sway back and forth in churning movements. A five-hour plate will invariably reveal duodenal and gastric residue and a dilated duodenum (Figs. 529, 531, and 533).

Disappearance of these findings by Hayes' maneuver is confirmatory. The Hayes' maneuver is as follows: Pressure is made by the hand over the abdomen just below the umbilicus. The mass of intestine is pushed upward, backward, and to the left, thus relieving the downward and backward traction on the mesenteric pedicle. In about thirty seconds the duodenojejunal angle is opened and the duodenal contents are seen to pass into the jejunum. The descending portion of the duodenum may be visualized in the lateral view. Hayes' maneuver is negative in duodenal obstruction caused by intrinsic or extrinsic tumors.

TREATMENT

The treatment of duodenal obstruction depends to a great extent upon the underlying causative factor. The treatment for the visceroptotic type and for the chronic form of duodenal obstruction is preferably medical. The objective in the medical treatment is to increase the fat content of the mesentery in order to elevate the small intestine upward and forward, thereby relieving the backward and downward traction on the duodenum by the mesenteric pedicle. This can be accomplished by postural rest in bed, high caloric fat and carbohydrate diet, and abdominal exercises. Having the patient rest and sleep in bed, the foot of which is elevated from 12 to 18 inches, permits the small intestine to migrate into its normal position. Also, the pressure on the duodenum may be materially relieved by having the patient lie on the abdomen as much of the time as possible, especially immediately after each meal (at least twenty minutes). The abdominal muscles are strengthened by abdominal massage, setting-up exercises, and alternate raising, lowering and raising of the legs at right angle with the body in the prone position. In the beginning these exercises must be done with moderation, and at no time must they tire the patient. At the same time the patient is placed on a nourishing diet. During the first few days the food intake should contain about 1800 calories, and gradually increased to 3000 calories. The major portion of the food should contain high caloric values in fats and carbohydrates, such as vegetables, fruits, cereals, fish, milk, cream, farinaceous puddings, butter, etc. The food should be thoroughly cooked and palatable, and small frequent feedings are preferable to the usual three meals a day.

Postural rest in bed, judicious exercise, and intelligent use of a high caloric diet prolonged for a period of months will bring about relief in the great majority of patients. If satisfactory results are not obtained, surgical intervention is indicated. The following criteria may be used to decide the question of operation: (1) failure of medical treatment to relieve the symptoms after prolonged fair trial; (2) inability of the patient to gain weight after three months of postural and medical treatment; (3) duodenal retention as shown by a five-hour *x*-ray plate; (4) persistent vomiting during medical treatment; (5) recurrent attacks of vomiting; and (6) when the cause of the obstruction is due to an intrinsic and extrinsic tumor.

CHOICE OF OPERATION

For the mesenteric type of obstruction the operation of choice is a duodenojejunostomy. This is the only procedure which adequately and thoroughly drains the obstructed duodenum. Some have suggested a *gastrojejunostomy; however, it* must be emphasized that this operation does not adequately drain the obstructed duodenum. It gives temporary relief by short-circuiting the gastric contents, but the duodenal contents are not emptied, and as a result, the patient may continue to complain of the original symptoms. A gastrojejunostomy may even aggravate the condition by promoting a deformity in the duodenal-jejunal angle, thus complicating the existing mesenteric obstruction. We agree with Wolfer (1927) that suspension of the ptosed viscera is uniformly unsuccessful for the cure of arteriomesenteric obstruction.

When the cause of the obstruction is an inflammatory or embryonal band, simple division of the band is the procedure of choice. Denuded surfaces must be peritonized by either an omental graft or by suturing, else the condition may recur. Gastrojejunostomy or better, gastroduodenostomy is the logical procedure in those cases with massive adhesions involving the first and second portion of the duodenum.

Colopexy, after the technique of Waugh, or colofixation to the anterior abdominal wall is the operation of choice for the mesocolic and arteriomesocolic obstruction. For the arteriomesocolic type, a duodenojejunostomy is a logical supplemental procedure, as colopexy or colofixation alone may not completely relieve the compression. Right colectomy as suggested by Bloodgood is too formidable and should be replaced by a more conservative and less dangerous procedure.

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CHAPTER 64

SURGERY OF THE SMALL INTESTINE

Consideration of Intestinal Obstruction

By A. V. Partipilo

INTRODUCTION

Murphy stated that for a period of thirty years prior to 1914 nothing of importance had been contributed toward the reduction of the mortality rate of intestinal obstruction. If Murphy were alive in 1925 he could have made the same statement by extending the period of inactivity for another decade. Since 1925 a prodigious amount of laboratory research and clinical observations have been made which has altered profoundly the management of intestinal obstruction. It is now known that the most serious consequences of an obstructed bowel are those due to loss of body fluid and electrolytes, and to gaseous distension. The necessity of supplying water and salt to correct chemical changes has been emphasized by a host of workers and is at present generally appreciated by the surgeon. The studies of Wangensteen and others have established the value of early and continuous decompression of a distended bowel. The application of these therapeutic measures have had a decided effect on the reduction of mortality rate, nevertheless it is still too high. The clinician and the surgeon must realize that when metabolic and chemical changes in the body have been greatly altered, the chances for recovery are diminished in proportion to the extent of these changes. Early recognition and prompt treatment are still the most important factors in the management of a patient with intestinal obstruction. For these reasons there should be no delay in the diagnosis, nor procrastination in operating.

DEFINITION OF TERMS

Intestinal obstruction, in its broadest sense, may be defined as an impediment, or hindrance to the forward passage of intestinal contents caused by intrinsic or extrinsic factors. The condition may be acute or chronic, complete or incomplete.

Ileus comes from a Latin word meaning to twist. At the present time the word is used to designate an obstruction due to paralytic conditions of the bowel. It is often used, however, interchangeably with the word obstruction; for instance, duodenal ileus or mechanical ileus. Both of these conditions are described more appropriately as duodenal obstruction or mechanical obstruction. To avoid confusion of terms, ileus should be used only for obstruction due dynamic or adynamic causes.

D[illegible]

inte[illegible]

from [illegible] conditions: (1) Local stimuli acting on the bowel at the site of the spasm; pinching, or squeezing the bowel during the course of an operation; the

presence of chemical, thermal or mechanical stimulants within the lumen of the bowel; and the presence of foreign bodies in the lumen. (2) Dynamic ileus may also occur as a result of a reflex spasm coming from extrinsic irritants, such as contusions of the abdomen, lesions of other organs, lesions of the coeliac axis, and lesions of the central nervous system.

Paralytic ileus has been defined as paralysis of the bowel due to peritonitis, the paralysis being the result of excessive stimulation of the splanchnics. Alvarez agreed with Hotz and Arai that in some cases of ileus due to peritonitis, the intestinal muscle is still capable of active contraction and is held back by nervous inhibition. He offered the explanation that there is a flattening or reversal of the normal gradient of forces and an excess of nervous inhibition. This is explained on the basis of the Flat-Gradient theory; namely, that irritation of the bowel at any point tends to hold back material coming down and tends to hasten the progress of material that has passed beyond the obstruction. It has been shown by Alton Ochsner and others that the most promising treatment of paralytic ileus is directed toward blocking the nervous inhibitory nerves (splanchnics) with spinal anesthesia.

Intussusception is a term applied to describe the invagination or the slipping of one part of the bowel into the part of the bowel continuous with it. It may be acute or chronic, the former being more frequent, and occurring during childhood. Intussusception may occur in any part of the intestinal tract, although the most frequent site is at the ileocecal valve.

Obturation generally refers to an obstruction produced by a material within the lumen of the bowel, such as gall stones, foreign bodies, hair-balls, etc.

Strangulation obstruction is produced by a compression of the intestine by a constricting band or by the neck of a hernial sac. The constricting ring interferes with the blood supply of the involved bowel with the result that gangrene of the bowel is the inevitable outcome of this form of obstruction.

By *compression obstruction* is meant an obstruction due to pressure from extrinsic causes, such as tumors, or enlarged or misplaced organs.

Stenosis or stricture indicates that the obstruction is produced by a contracture of the musculature or from scar tissue formation.

Coprostasis is an obstruction due to fecal impaction, while *obstipation* refers to an obstinate or intractable constipation.

Constriction is a term employed to describe an obstruction due to a fibrous band, adhesions, or hernial sac.

Simple obstruction means that the obstruction is not attended with bowel changes. *Gangrenous obstruction* denotes that the involved bowel has circulatory disturbances with gangrene of its wall.

High obstruction refers to obstruction of the bowel above the ileocecal valve, while low obstruction is below the valve.

Volvulus may be defined as an obstruction due to twisting or knotting of the intestine. It is most frequently seen in the sigmoid flexure of the colon.

CLASSIFICATION OF INTESTINAL OBSTRUCTION

Etiological classifications for disease entities are, as a general rule, inadequate and often confusing. This is no less true with similar classifications of intestinal obstruction. Various names as applied describe the causative factors, with the result that confusing terms are used to describe similar conditions.

A classification based upon pathological changes in the bowel wall would go far in clarifying the subject. It would also be ideal for purposes of discussion and study. From this standpoint then, intestinal obstruction may be classified into two general groups: (1) simple obstruction with no circulatory involvement and no damage to the bowel wall; and (2) gangrenous obstruction.

SIMPLE OBSTRUCTION

Simple obstruction occurs in the fixed sections of the intestinal tract, such as the pylorus, duodenum, ascending and descending colon, and the pelvic colon. The inevitable result of simple obstruction of the jejunum and ileum is gangrene of the involved segment of bowel. This is due to numerous folds of mesentery and coils of intestine which predispose to circulatory changes. For this reason any obstruction involving the jejunum and the ileum should be considered at the outset as gangrenous obstruction.

From an anatomical point of view, simple obstruction may be divided into two groups: (1) high simple obstruction occurring above the iliocecal valve, and (2) low simple obstruction occurring below the iliocecal valve.

High Simple Obstruction.—As previously stated high simple obstruction occurs above the iliocecal valve. Conditions which may cause obstruction in the pyloroduodenal region are: (1) gastric and duodenal ulcers producing either an acute pylorospasm or cicatricial stenosis; (2) gastric neoplasms causing stenosis of the pyloric sphincter; or (3) obstruction resulting from a pedunculated tumor which has passed through the sphincter, thereby preventing the stomach from emptying itself. The pyloroduodenal region may also become compressed by an enlarged biliary calculus, by adhesions associated with cholecystitis, or from a stone which has perforated the duodenum. In the latter instance the history is that of an acute duodenal obstruction engrafted upon a previous history of cholecystitis and cholelithiasis. Conditions producing obstruction of the third portion of the duodenum are dealt with in Chapter 63.

The primary effect of a high obstruction is an early dehydration, as a result of vomiting and disturbance in the absorption of fluid. Due to the loss of fluids, the blood viscosity is increased, which in turn embarrasses the circulation. Dehydration is manifested by a dry and inelastic skin and parched mucous membrane. The urine becomes scanty, highly colored; later it will contain casts, albumen, and eventually blood. These and other factors which will be discussed shortly, readily prove fatal unless treatment is promptly instituted.

The cardinal symptom of high intestinal obstruction is vomiting, and the higher the seat of the obstruction the earlier this symptom appears. In pyloric stenosis the vomiting is regurgitant in character, continuous, and copious in amount. Pain is a variable factor; the patient is often relieved after the stomach is emptied. In duodenal obstruction the vomiting is intermittent and the vomitus contains enormous quantities of bile. Pain may be severe during the attack because of the unyielding spastic pylorus. When the latter gives way, the patient vomits and is relieved of pain.

It is a well-known fact that dilatation of the bowel occurs proximally to an obstruction, whereas distally, it is collapsed. Hence, in obstruction above the duodenal-jejunal flexure, epigastric fullness is the usual finding, rather than abdominal distention.

The cardinal symptoms and signs of obstruction of the jejunum and ileum are: (1) pain; (2) abdominal distention; (3) vomiting; (4) constipation; and (5) visible peristalsis. The outstanding feature in obstruction of the small intestine is the abdominal pain, which is colicky in nature and is associated with progressive distention of the abdomen. This is in contrast with pyloric or duodenal obstruction where *vomiting* occurs early and epigastric fullness is present rather than abdominal distention. The abdominal pain comes on suddenly and the patient can tell the very hour when the "cramps" began. It is generalized over the entire abdomen, and it is severe and colic-like. The pain reaches the maximum of severity in about two minutes, then it subsides for a variable interval and recurs again and again. At the height of pain, bubbling or gurgling sounds may be heard over the abdomen with a stethoscope. *This is pathognomonic of intestinal colic of an organic nature.* According to Wangensteen, intestinal obstruction of mechanical origin without intestinal colic does not exist. Therefore, the first step in determining the absence or presence of mechanical bowel obstruction is to decide whether intestinal colic is present. Concomitant with intestinal colic is the finding of abdominal distention. It is progressive, and marked distention is a grave and late sign. One should not wait until the abdomen is markedly distended to arrive at a correct diagnosis.

Vomiting in obstruction of the ileum or jejunum is at first due to a reflex mechanism. Later, some hours after the onset of pain, it is constant and abundant. Obviously, the higher the obstruction, the earlier it will appear. The fecal odor indicates that the content of the lower reaches of the obstructed bowel has been contaminated with colon bacilli from the cecum, and is not indicative of large bowel obstruction. A patient with obstruction above the ileocecal valve may have one or two bowel movements soon after the onset of distress, however, once the bowel below the obstruction empties itself, there is absolute constipation. Neither gas nor fecal matter is passed and the patient is very much aware of this. In thin individuals, and in the early stages of obstruction, visible peristalsis may be seen. In the obese, or rigid and distended abdomen, this sign is obscured. It is an early sign of obstruction.

The *diagnosis of a high intestinal obstruction* should be based upon the acute onset of intestinal colic associated with borborygmi, progressive abdominal distention, vomiting and constipation. Analysis of the sequence in development of these symptoms and signs will invariably reveal the site of the obstruction. A patient with a high intestinal obstruction will give a history of sudden cramp-like pain occurring at a definite hour, followed by vomiting and abdominal distention and later constipation. The characteristics of a low obstruction are: abdominal distention, definite constipation, abdominal pain, and very late vomiting if at all. Vomiting is the first sign of obstruction of the third portion of the duodenum, and the vomitus contains enormous quantities of bile; whereas, vomiting of clear fluid is indicative of pyloric obstruction. In obstructions above the duodenal-jejunal flexure, intestinal colic is absent and the abdomen is not distended, although there is epigastric fullness. Vomiting immediately following an abdominal operation may be due to compression of the terminal duodenum by the mesenteric pedicle. *Placing the patient on the abdomen, or in the knee chest position, will often correct the condition.*

Low Simple Obstruction.—The most common cause of obstruction of the large bowel is malignancy. Ulceration of the colon followed by cicatricial contraction is

not an uncommon cause. Other causes are fecal impaction, impaction of a foreign body, diverticulitis, and, more rarely, congenital strictures of the rectum.

In obstruction of the large bowel the acuteness and severity of the symptoms depend upon the location of the lesion and the nature of the causative agent. Diseases of the right colon rarely cause acute obstruction because its content is in a state of fluidity, whereas in the left colon acute obstruction is more common because the fecal content is normally formed, thus requiring a lesser degree of stenosis to produce obstruction. The presence of normal angulations in the hepatic and splenic flexures favor acute obstruction, especially in the splenic flexure where acute obstruction takes place in almost every case.

Patients with acute obstruction of the large bowel may present the same clinical symptoms and findings occurring in small bowel obstruction, however, the progress of the disease is slower and the symptoms are less severe. Abdominal distention is more pronounced and more diffuse, while vomiting occurs rather late. The ileocecal prevents regurgitation of gases and fluids from the large bowel into the small intestine. For this reason a patient with obstruction of the large bowel may not vomit until very late in the disease. Constipation is present in all cases of complete obstruction, however, the higher the obstruction the more likelihood of bowel movements to occur after the onset of the symptoms. On the other hand, constipation assumes a prominent and significant role the lower the seat of the obstruction. In rectal carcinoma irregularity of bowel movements and constipation are outstanding features. The patient may complain of attacks of constipation and diarrhea for months before the onset of pain and gaseous distention. Vomiting in rectal obstruction is a rare and terminal symptom.

The use of the flat roentgen-ray plate of the abdomen is an invaluable aid in the diagnosis of intestinal obstruction. Interpreted in the light of clinical findings it will reveal (1) the presence of an obstruction; (2) whether it is complete or incomplete; (3) whether the large or small bowel is involved; and (4) will determine the position of an indwelling tube.

The film should be made with the patient lying on the back. Under normal conditions, a flat plate of the abdomen will show gas shadows in the stomach and colon, but none in the small intestine. This is due to the fact that gases become separated from colonic contents, whereas, in the small intestine they are so thoroughly mixed with the juices that none is visible on the x-ray plate. Therefore, a normal plate will not visualize the intestine. The characteristic evidence indicating obstruction of the small intestine is the finding of outlines indicating dilatation and the presence of parallel lines crossing the bowel transversely. The parallel lines give a "herring-bone" appearance and are due to the folds of Kerkrind which stand out while the rest of the mucous membrane is thinned out by the pressure of the gas. The outlines of the intestine are in step-ladder arrangement. Whether the obstruction in the small intestine is complete or incomplete is determined by noting the absence or presence of colonic gas after a preliminary evacuant enema has been administered. If the x-ray plate shows distention of the small bowel and absence of gas in the colon after the preliminary enema, the obstruction is complete. Distention of the small bowel and gases in the colon, is indicative of incomplete obstruction. In obstruction of the large bowel, a roentgen film shows no distention of the small intestine, but the colon is considerably distended, especially the cecum. The haustral markings are visible, but they do not run all the way across the bowel, and the distance between them is increased.

GANGRENOUS OBSTRUCTION

As previously stated, gangrenous obstruction occurs in the movable sections of the intestinal tract. This is due to numerous folds of mesentery and coils of intestine, which, under certain circumstances, favor circulatory changes in the bowel wall. What are the factors and mechanisms in the production of gangrene and circulatory disturbances in acute intestinal obstruction? They are: (1) gaseous distention; (2) distention of the bowel proximal to a constricting band; and (3) direct vascular changes.

Gaseous Distention.—Every surgeon is familiar with the difficulty of maining distended loops of bowel within the abdomen after an incision has been made. This distention must impress one with the tremendous pressure existing within the lumen of the bowel. There have been cases reported of bowel perforation as a result of gaseous distention. Undoubtedly, it is the most dangerous factor in the production of gangrene. In 1898, Kocher announced that gaseous distention alone can cause gangrene; others have since proved by actual measurement the amount of intra-intestinal pressure necessary to produce arterial stasis. Gatch and his coworkers found by actual experiments that intestinal distention causes a decrease in the blood flow through the bowel wall in direct proportion to the elevation of the pressure. Blood flow almost stops when the intestinal pressure reaches the level of the diastolic pressure. When this occurs the mucosa becomes devitalized within five to fifteen minutes. They found that when the mucosa is devitalized, toxic substances are absorbed. In obstruction, stasis is enhanced by the lowering of the blood pressure due to loss of fluids, and by the increased viscosity of the blood. The greater the fall in blood pressure and more viscid the blood becomes, the amount of intra-intestinal pressure required to obliterate the blood supply is proportionately lessened. This must be taken into consideration in the treatment; the blood pressure must be maintained, and the viscosity of the blood decreased.

Distention Proximal to a Constricting Band.—The second factor in the mechanism producing gangrene is distention of the bowel proximal to the obstruction, thereby exerting an enormous traction against a constricting band. It is because of this traction rather than the presence of a constricting band that gangrene occurs. The band must have been present long before gangrenous changes take place, hence it is the action of gas distention which produces the circulatory changes. In this type short loops are involved. Foster believes that in these cases toxemia is the primary cause of death. This should be kept in mind during the operation, because if this highly toxic material is spilled into the peritoneal cavity death quickly follows.

Direct Vascular Disturbances.—The third factor in the production is a direct interference in the vascular supply to a segment of intestine. This occurs in mesenteric thrombosis, volvulus, intussusception, and strangulated hernia. In all cases, gangrene is the result of either a compression, a twist, or thrombosis of the vessels supplying a segment of bowel. However, it is obvious that in certain conditions, increased intra-intestinal pressure influences the process. This is especially true in strangulation and in obstruction due to compression.

EFFECTS OF OBSTRUCTION

The primary effect of high intestinal obstruction is dehydration as a result of continued loss of fluid in the intestinal secretions of the distended bowel and from

vomiting. As a result of vomiting there is also a loss of chloride of the blood. This [illegible] that the recoveries occur more often in cases when the blood chlorides are not greatly altered, and in cases with a low count, who are treated with adequate amount of saline solution. It has also been observed that the fall in chlorides is greater in the high obstruction than in the low.

With the loss of chloride the base is set free to combine with the carbon dioxide to form sodium and potassium carbonate. This is an attempt to equalize the electrolytic balance. With continued loss of chloride there is an increase in the carbon dioxide combining power of the plasma thereby giving rise to alkalosis. This condition will predominate as long as the non-protein-nitrogen remains within normal limits. However, with retention of sulphates and phosphates, and as the N-P-N rises, the reaction of the blood may shift from the level of alkalosis to acidosis. The rise in the carbon dioxide combining power somewhat parallels the fall in chlorides, but the relationship is not constant since other factors are operative.

With continued loss of chloride the sodium radical is eventually lost by being excreted in the urine. This further disturbs the water balance, since sodium is an important factor in the maintenance of water in the tissues. Thus, with loss of sodium the interstitial reservoir of fluid is severely compromised and the patient shows signs of well advanced dehydration. As a result, the viscosity of the blood is increased as manifested by hemoconcentration and reduced blood volume. A moderate degree of concentration leads to impairment of the circulation. This is due to the inability of the sticky blood to pass through the capillaries. If the process is permitted to continue, the body tissues become asphyxiated and finally cellular death eventually takes place.

Effect of Gaseous Distention.—*The effects of gaseous distention* are manifested locally upon the bowel wall. The impairment of circulation of the bowel wall from intra-intestinal pressure has been discussed. This is the most serious consequence of distention. In addition there is an interference in the normal lymphatic capillary function, with the loss of selective power of absorption. Singleton, quoting Gatch, believes that this is due to (1) diminished blood flow through the intestinal wall; (2) altered physical forces involved in absorption; and (3) damage to the intestinal wall which affects both the activity of the mucosa and the physical forces which hinder or promote absorption. Loss of the power of absorption permits accumulation of enormous quantities of fluid. Under normal conditions, eight to ten liters of fluids are poured into the upper part of the gastro-intestinal tract and practically all of this is reabsorbed in the lower intestinal tract. But, in obstruction, practically all of this fluid is lost and the condition is further aggravated because of the increase in the excretion of fluid as a result of distention.

Additional effects of distention are: (1) interference in the motility of the bowel; (2) edema of the bowel wall: (3) stretching eventually cause atony of the bowel; (4) damage to the mucosa with possible resulting increase in the absorption of toxic materials; (5) there is evidence of plasma loss due to distention of the bowel; and (6) the effects of distention on the liver, kidney, and the heart and lungs through pressure on the diaphragm.

CAUSES OF DEATH

Numerous opinions have been expressed concerning the cause of death in intestinal obstruction. Many have come to diverse conclusions, while others have

agreed to several factors. McIvers believed that dehydration with loss of electrolytes, phosphates, and carbonates was the main cause. Gatch supported him and added to this the loss of chlorides and starvation. Many investigators have shown the important role that dehydration plays in producing metabolic disturbances in general. In his studies of tissue changes associated with acute loss of body water, Davis has shown that there is wide spread distention and increase in number of open capillaries throughout all the tissues. These changes are correlated with conditions associated with hemoconcentration and the final effect of this is a deficient oxygenation and anoxemia. Rubner found that in starvation an animal can lose practically all of its glycogen and fat, half of its body protein, 40 per cent of its total body weight, and still live, whereas the loss of 10 per cent of water content results in serious disorder, and the loss of from 20 to 22 per cent results in death.

The importance of afferent nerve impulses from distended intestine as a direct factor in causing death has been emphasized by many workers. Herrin and Meek showed by animal experimentation that denervation of an obstructed loop of intestine protected an animal against death for an indefinite period. Other workers were not able to prolong life by denervation of the distended loop. Dragstedt demonstrated that the cause of death and morbidity of high intestinal obstruction was due to failure of re-absorption of water and salts secreted in the gastrointestinal tract. Taylor and his associates believed that distention was the primary cause of death and Wangensteen has shown that regardless of fluid and salt replacement, life is not maintained over long periods of time unless acute distention of the intestine is relieved. The effect of loss of plasma into the wall of the distended proximal intestine as causing a depletion of circulating volume and death is held by many. Fine and Gendel showed that loss of plasma continues as long as distention continues and that the extent of this loss, if uncompensated, is sufficient in itself to cause death. Scudder and his coworkers believed that in simple obstruction, a rise in blood potassium occasioned by loss of fluids is a factor in the cause of death. They suggested that the elevation of blood potassium is associated with adrenal insufficiency. Others believe that toxemia is the important lethal factor in the cause of death and consider this in the treatment. For instance, Holden believing that the contents of an obstructed bowel are toxic, mechanically emptied the entire intestine proximal to the obstruction in every case. He reported a marked reduction in the mortality rate as a result of this treatment. In spite of this, one cannot help but feel that routine evisceration and drainage is a dangerous procedure.

From a study of the recent literature, it must be concluded that the cause of death in simple obstruction is not due to any one single factor, but to a combination of physiological disturbances which to date are not fully understood. In many cases the determining cause of death may depend upon the age and body resistance of the patient, and whether or not associated cardio-renal disease complicates the process. In high obstruction the loss of chlorides and the effects of dehydration and distention are undoubtedly the main causes, whereas, in low obstruction inanition and altered protein metabolism are the important causes. Nevertheless, in both the high and low obstructions, metabolic and blood changes are present and the difference in the severity of the effects and symptoms depend upon the physiological function disturbed in the various sections of the gastrointestinal tract.

Many investigators believe that the cause of death in gangrenous obstruction is due to a toxemia resulting from absorption of toxins. The nature of the toxins is not definitely known. Whipple and his associates thought that the toxin was a

proteose, a perverted secretory product of the epithelium of an obstructed bowel. Others claimed that the toxin was the result of the action of the pancreatic enzymes on the protein within the stagnant bowel. Other investigators claimed that the toxin is similar if not identical to histamine. In the experimental laboratory it was found that injection of material found in the intestine of an animal suffering from obstruction produced symptoms similar to histamine poisoning.

With due consideration to the above theories, bacterial infection and toxemia cannot be disregarded; especially since the contents of stagnant bowel forms a good culture medium. Experimental evidences by Blain and others indicates that bacteria play an important role in death from strangulation obstruction. Blain has also demonstrated that the lethal effects of bacterial growth can be significantly obviated by massive doses of penicillin. The nature of the toxin nor the exact mechanism by which bacteria produced their lethal effects have not been explained.

To summarize the foregoing statements, the following conclusions are made: (1) That the cause of death in simple obstruction is due to fatal alterations in metabolic and body chemistry resulting from vomiting and to distention of the bowel. (2) Life is compatible over a long period of time in simple obstruction providing replacement therapy with salt and water is maintained and the bowel is kept deflated. (3) Gaseous distention is the most serious and vital factor in the cause of death in both the simple and strangulation obstruction. (4) Death occurs relatively early in gangrenous obstruction. This is probably due to the absorption of a toxic substance of unknown origin. Clinical experience has shown that the spilling of the contents of a gangrenous bowel into the peritoneal cavity causes death in almost every instance, whereas this is not true in cases of simple obstruction. (5) Gangreous obstruction is incompatible with life unless the involved segment is removed early and the proximal contents adequately drained.

TREATMENT

Early Diagnosis.—Notwithstanding the advancements of recent years in the treatment of intestinal obstruction, the mortality rate is entirely too high. We believe that this is due partly to failure to realize that every case of simple obstruction of the small intestine is a potential gangrenous obstruction and that the only treatment of value for the latter condition is early operation. Thus, we have come to the conclusion that the most important single factor in the reduction in the mortality rate in acute simple mechanical intestinal obstruction is to operate early. It is an obvious truism that to operate early the condition must be recognized early, before fatal changes take place. If the diagnosis can be made without loss of time it is to the glory and credit of the attending physician, however, procrastination over a diagnosis does not help the patient. When in doubt as to the viability of the bowel, it is good judgment to make a diagnosis of a "surgical abdomen" as an excuse to investigate rather than wait until the patient is moribund. In this manner an operative diagnosis is made, which is preferable to a postmortem one. Very often, the blame for the delay rests upon the attending physician. On the other hand, he is not the only one to blame, for the surgeon falls into the same error. Statistics show that the surgeon rarely institutes early operative treatment of acute postoperative obstruction. In this connection C. H. Mayo in 1922 stated, "Obstruction following operative procedures is a real tragedy. While we blame the patient and the medical attendant for delay in calling counsel in the obscure cases, the same

delay occurs in hospitals in postoperative cases, and there is a lack of promptness of action, because of the hope the obstruction is partial and will 'clear up tomorrow.'"

Another cause of delay is the promiscuous use of morphine which relieves pain, but masks the important symptoms. Narcotics should not be *given to relieve pain* until a diagnosis has been made. Nor should they be used when the patient or relatives refuse consent to operation. It is for the well-being of the patient that he suffer rather than have a few hours of comfort and eventual death.

The next important step in the management of intestinal obstruction is to evaluate the extent to which the chemical and metabolic processes have been altered and to treat these and the associated distention as thoroughly and early as possible. Thus, the medical treatment is directed toward the following objectives: (1) relief of dehydration; (2) restoration of altered chemical changes; (3) restoration of plasma level to normal; (4) combat any bacterial process that may be present; and (5) decompression of the distended bowel.

Fluid and Electrolytic Balance.—The amount of fluid and salt required to combat dehydration and hypochloremia depends on the duration and severity of the obstruction. The degree of dehydration may be determined by various ways, such as, (1) by evaluating the clinical syndrome of dehydration; (2) hematocrit readings; (3) erythrocyte count; (4) hemoglobin determination; (5) plasma protein level; (6) plasma chloride determination; (7) amount of urinary output; and (8) quantitative measurements of intake and output (see Chapter 2).

According to Maddock and Coller, a patient weighing 60 Kg. who shows signs of severe dehydration requires during the first twenty-four hours about 7000 cc. of fluid as given in Table 9. This is a great deal of fluid, but these investigators have

TABLE 9.—Water requirements in a dehydrated patient weighing 60 Kg.

1. Water for vaporization	2000 cc.
2. Water for urine	1500 cc.
3. Abnormal loss, if any, during the 24 hours	....
4. Water to restore depleted fluids, 6 per cent of 60 Kg.	3,600
Total	7,100 cc.

found that such quantities are necessary to provide for dehydration, for vaporization and abnormal loss, and then have enough fluid left for kidney function. Once the depleted fluids have been replaced, much less fluid is needed to maintain water balance. The simplest guide of determining subsequent fluid requirement is to measure the urinary output. With normal functioning kidneys a daily urinary output of 700 to 1000 cc. is a good indication that the patient is receiving sufficient fluid.

Patients manifesting milder degree of dehydration require about 3500 cc. of fluid. It supplies 2000 cc. of water for vaporization and 1,500 cc. for excretion by the kidneys. *This is the amount of fluid required by any seriously ill patient.* For the obstructed patient, additional fluid should be given to replace that lost by vomiting or by suction drainage.

Coller and Maddock stress the importance of the kidneys in maintaining water balance. If there is plenty of available water, the urinary wastes are put out in a large volume of urine of moderate or low specific gravity. If the water is scarce, the urine volume is small and its specific gravity is high. It must be emphasized that a small urinary output of high specific gravity means that the patient is receiving insufficient amount of fluid. Although a comparison of the fluid intake and

output gives an easy and fairly accurate method of determining the patient's water balance, a complete picture of the total water exchange is obtained by checking the plasma protein level, hematocrit, erythrocyte and hemoglobin determinations, and determination of plasma chlorides. In this manner, abnormal conditions are detected. For instance, administration of excess sodium chloride in the face of an impaired renal function will result in retention of the salt, and, if permitted to continue, consequent tissue edema. This can be averted by checking plasma chloride levels and hematocrit determinations. Fine and his associates have shown that patients with distention of the small intestine show a considerable loss in the volume of circulating plasm, and as the plasma volume falls the hematocrit rises. This increase in hematocrit is far greater than can be explained by dehydration, however severe. Thus, plasma and hematocrit determinations are essential for reliable information as to the true picture of the patient's state of hydration. Also, they indicate the necessary corrective therapeutic measure.

The degree of dehydration having been established, the next important task is to determine the kind of fluid to give to restore the altered blood chemistry. The administration of saline solution is indicated to restore the depleted chlorides of the blood. A dependable method of determining the amount of saline to be given has been determined by Coller and Maddock. Patients with depleted water and electrolytes as a result of fluid loss previous to admission to the hospital, the following clinical rule was applied; "for each 100 milligrams that the plasma chloride level needs to be raised to reach the normal (560 milligrams per cent), the patient should be given 0.5 gram of salt per kilogram of body weight." Examples of the use of this clinical rule in hypothetical cases are given in Table 10. Experience has

TABLE 10.—[illegible]
Maddoc [illegible]
normal

Weight Kg.	Initial plasma chloride level mgm. per cent	Calculation
60	460	1.0 by 0.5 by 60 = 30.0 gm.
60	410	1.5 by 0.5 by 60 = 45.0 gm.
50	500	0.6 by 0.5 by 50 = 15.0 gm.
70	360	2.0 by 0.5 by 70 = 70.0 gm.
45	440	1.2 by 0.5 by 45 = 27.0 gm.

shown that giving more salt than that calculated from the clinical rule is useless and that it only aggravates the normal distribution of fluids. The required salt is given as a physiological solution or Ringer's solution. Ringer's solution is preferred because there is less abnormal retention of water when it is used. Ringer's solution contains approximately 9 grams of sodium chloride per liter. To calculate the amount of solution needed to correct the hypochloremia, the amount of sodium chloride is first determined by the clinical rule, and then, the calculated amount of salt is given as Ringer's solution. Additional fluid required for vaporization and water for urine may be given as 5 per cent dextrose in distilled water. This amount of salt and glucose solution is given during the initial treatment of a severely dehydrated and hypochloremic patient, and the total amount of fluid may be spread over a period of forty-eight hours. After this initial replacement therapy, the amount of salt need not be greater than that required by a normal individual (2 to 10 grams), and additional fluid for water of vaporization (2000 cc.) and water for

urine (1500 cc.) is given as 5 per cent glucose. If the patient is losing large amounts of fluid as a result of gastrointestinal drainage, it is essential that this amount is replaced volume for volume with physiological or Ringer's solution. The use of glucose with water and salt has several advantages. Besides offering an immediate source of energy, it provides carbohydrates for glycogen formation and for oxidation of ketones if they are present.

Plasma and Nutritional Balance.—One of the undesirable features of high intestinal obstruction is the acute loss of plasma and the associated hypoproteinemia. Acute loss of the water or protein fraction, or both, results in a reduction of the plasma volume, hemoconcentration and an increase in the carbon dioxide content of the blood. Fine and his associates have shown that his fall in plasma volume occurs even when fluid and electrolyte imbalance is prevented. A deficiency in the protein fraction produces a disturbance in the delicate mechanism in maintaining a state of balance between the blood and the intercellular fluid, caused by a decrease in the colloidal osmotic pressure of the blood. The relationship between hypoproteinemia and tissue edema has been well established. Ravdin and others have shown that disturbances in the motility of the gastrointestinal tract, wound disruption and evisceration are due to edematous tissue secondary to a deficiency in the protein fraction. According to Efskind, the relative limit at which the occurrence of edema may be feared lies at 5.6 per cent of total protein. The absolute limit, below which edema invariably appears lies at 4.5 per cent. It must be emphasized that latent edema may occur at protein levels close to normal percentages.

It is quite obvious that an estimation of the extent of loss of plasma proteins is of considerable value and that early correction of protein deficiency and the decreased plasma volume is imperative to prevent irreversible changes. Exact protein requirement can be calculated by determining the nitrogen balance, however this is not practical. Upon admission to the hospital, a satisfactory approximation of the amount of protein required may be estimated by the patient's clinical history, i.e., (1) the length of time the patient has been obstructed, (2) the degree of dehydration, (3) the severity of the distention of the bowel, and (4) adding to these estimated deficiencies one gram of protein per kilogram body weight. Subsequent protein requirement is determined by plasma protein level, hematocrit, and erythrocyte count.

For the severely obstructed patient showing signs of serious dehydration, replacement therapy is directed toward increasing the osmotic colloidal pressure, overcome hemoconcentration, and increasing the circulating blood volume. This is in addition to the correction of the electrolytic imbalance and dehydration with salt and glucose. During the initial treatment of the seriously dehydrated patient, salt should be given with great caution. It may actually decrease the circulating plasma by washing it out of the blood stream into the interstitial spaces. Nor is whole blood indicated in the presence of hemoconcentration and increased viscosity of the blood. The most efficient solution is plasma. For the seriously dehydrated patient requiring 7000 cc. or more, of fluid during the first forty-eight hours, as much as 2000 cc. of this fluid can be given as plasma; the remaining portion (5000 cc. plus) is given as 5 per cent glucose and salt solution. The salt solution is estimated according to the clinical rule of Coller and Maddock as previously discussed.

Intestinal Decompression.—The next important problem in the management of intestinal obstruction is to relieve the intestinal distention. Regardless of replacement therapy, early relief of distention is of the utmost urgency. Decom-

pression may be accomplished by enterostomy or intubation. Decompression by enterosotomy was the procedure of choice until about 1933 when Wangensteen and his co-workers introduced and popularized the principle of decompression by suction applied to an indwelling duodenal tube. With the introduction of the Miller-Abbott long tube which permits decompression at the site of the obstruction, there is less indication for enterostomy. Nevertheless, in some cases enterostomy may be justified.

The underlying principle of the moden methods of decompression is to introduce a tube into the gastrointestinal tract for the purpose of removing gases and fluid accumulated in an obstructed bowel. Gastric drainage and suction was first suggested in 1925 by Ward, and later Wangensteen advocated continuous duodenal suction. It was early recognized that these methods drained the distended bowel at a point remote from the site of the obstruction, thus removing a much greater quantity of fluid than if the bowel had been drained near the point of the obstruction. The Miller-Abbott double lumen tube overcomes this objection by draining the bowel close to the obstruction rather than at the top. Hence, the Miller-Abbott tube accomplishes decompression similar to an enterostomy. It is also useful in that oral nourishment can be given while suction is being applied.

The Miller-Abbott tube is 10 feet long and 16 French in diameter. It is a double lumen being separated by a septum throughout its entire extent. The inflation tube opens into a soft rubber balloon and the suction end has several openings and terminates in a metal tip. The well lubricated tube is passed through the nostril into the posterior pharynx. The patient is then made to swallow water and the tube is gently guided into the esophagus and stomach until the 75 centimeter mark is reached. The patient is now placed on the right side and this position is maintained until the tube has passed into the duodenum. This may take from two to six hours. The most frequent cause of failure of the Miller-Abbott tube is the inability to pass the tube in the duodenum. Therefore, it is important to know if the tube has passed the pylorus. Wise gives three aids in determining this: (1) The syringe test; an empty 30 cc. glass syringe is used to inject 30 cc. of air into the balloon. If the balloon is in the duodenum the peristaltic action of the intestine will constrict the balloon and cause the plunger to be pushed back for a short distance. A minute later the plunger will be pushed back for a short distance. If the tube remains in the stomach no movement is seen or is pushed back steadily for its entire length. It is important to use a syringe whose plunger moves in and out easily. (2) The character of the fluid drained may be of value; the stomach contents are colorless while the duodenal fluid is deeply biled stained. When obstruction has existed for a long time, the contents of the stomach and duodenum are identical. (3) *X*-ray is the most definite method of determining the position of the tube. After the tube has passed into the duodenum, the balloon is inflated with 30 cc. of air and the balloon tube is clamped. Wangensteen suction is now applied and the tube is permitted to move 6 inches every hour until the 8 foot mark is reached. The suction tube is irrigated each hour with 20 cc. of water.

To overcome some of the mechanical disadvantages of the original Miller-Abbott tube, many modifications have been made. Of these, the most practical one is that devised by Harris. This consists of a single lumen mercury weighted tube. The principles upon which the Harris tube is predicated is that of a weighted bag carrying the tube down into the small bowel by force of gravity, in contrast to the principle of peristaltic activity grasping the inflated bag of the Miller-Abbott

urine (1500 cc.) is given as 5 per cent glucose. If the patient is losing large amounts of fluid as a result of gastrointestinal drainage, it is essential that this amount is replaced volume for volume with physiological or Ringer's solution. The use of glucose with water and salt has several advantages. Besides offering an immediate source of energy, it provides carbohydrates for glycogen formation and for oxidation of ketones if they are present.

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tube. The fluidity of the mercury molds it to the outline of the nasopharynx, esophagus and pylorus, resulting in minimum discomfort and trauma arising from its passage. The Harris tube is a flexible rubber tubing with a caliber of 14 French, and 10 feet long. Perforations are present along the first foot of the tube for aspiration when suction apparatus is attached. Two metal sleeves are fitted into the nasal end of the tube, one about two centimeters above the tip, the other 12 centimeters distal to the first. A thin rubber bag or condom 12 centimeters long is used to hold the mercury in place on the tube (Fig. 534). A hole is made in the

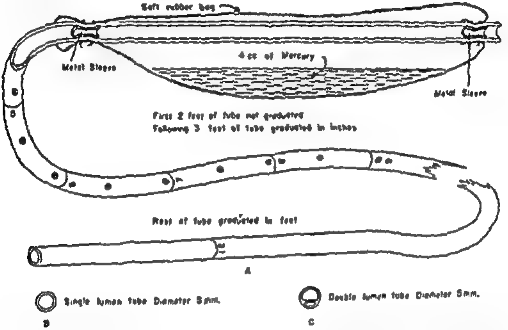

FIG. 534.—Mercury filled bag tied to Harris tube. Note metallic sleeves inset in lumen at points corresponding to tied [illegible] tube. (b) Cross section of single lumen tube, showing entire [illegible] tube diameter is less than that of (c) the Miller-Abbott tub[illegible] Gynec. & Obst.)

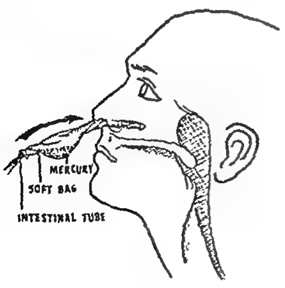

FIG. 535

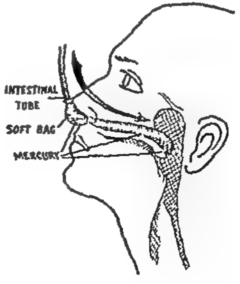

FIG. 536

FIG. 535.—Single lumen mercury weighted intestinal tube in position for insertion. Note mercury in proximal end of twisted lubricated bag.

FIG. 536.—Tube is elevated in order to allow mercury automatically to flow into empty nasopharyngeal end of bag.

closed end of the bag which is then inverted, slipped over the tip of the tube for a distance of about two centimeters and securely tied with No. 1 black silk thread The bag is then pulled back over its fixed end, 4 to 6 cubic centimeters of metallic mercury are placed in it, and its free end is securely tied to the tube. By fastening each end directly over its corresponding metal sleeve the tube is prevented from collapsing due to the pressure of the ligatures.

Technique in the passage of the Harris tube is as follows: It is imperative that no air is in the mercury filled condom. The bag is wrapped around the tubing with the mercury at its proximal portion. As illustrated in Figure 535, the glycerine lubricated tube is introduced into the naris. If the patient is unusually apprehensive, local application of cocaine may be required. After insertion of two inches, the tube is elevated (Fig. 536) to permit the mercury automatically to flow into the distal part of the bag, thus aiding the passage of the tube into the nasopharynx (Fig. 537) and down into the stomach. After the tube has passed into the stomach,

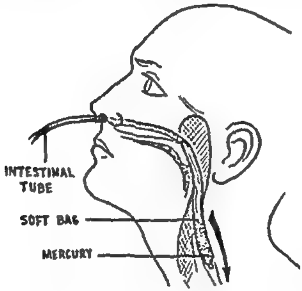

Fig. 537.—Due to weight of mercury, tube has descended into esophagus; bag is untwisted.

Wangensteen suction is applied and the contents of the stomach are completely emptied. The patient is now placed on the right side in the semi-Fowler position. If the patient can stand up or even walk around for five to ten minutes or longer, a more rapid descent of the tube is obtained. The following details of technique must be adhered to if smooth progress of the tube is to be accomplished: (1) see that the progress of the tube is not impeded by adhesive tape attached to the neck; (2) allow a slack of 6 to 12 inches along side of the naris so that spontaneous descent is observed; (3) after the "inch mark" reaches the naris, the tube must not be permitted to move spontaneously or to be helped along at a greater rate than 1 inch every ten minutes. Too rapid descent of the tube invariably results in coiling of the tube in the stomach, thus preventing its passage into the duodenum. The nurse on duty should be informed of this technical detail; (4) *x*-ray follow-up. During the first forty-eight hours, at least three films should be taken at twelve to eighteen hours apart.

The danger of mercurial poisoning in cases of rupture of the bag is one of the theoretical objections to this technique. Harris has shown by laboratory and clinical material that there is little if any danger to the patient should the mercury become free in the intestinal tract.

The introduction of intubation in the treatment of intestinal obstruction has been one of the outstanding advancements within recent years. When properly applied, it offers a simple and convenient method of deflating the bowel and removing stagnant and toxic material. When surgical interference is indicated, preoperative decompression permits greater freedom of manipulation and allows the point of obstruction to be reached with greater ease. The *rationale* of intubation as a method of producing decompression is now well established, however, the underlying reasons for its use in the various types of obstruction are not as thoroughly established. From a clinical point of view, the indications for intubation may be classified into three groups; (1) paralytic or adynamic ileus and simple obstruction of mechanical origin. In this group of cases intubation alone is especially indicated. The mechanism by which decompression is accomplished in a dynamic ileus is explained by Crowley and Johnston as follows: The tube is passed by the active bowel to the inactive segment where there is insufficient peristalsis to carry it further. At this point the accumulated gas and fluid in the inactive segment is withdrawn and further accumulation is prevented. Reduction of distention and prevention of its recurrance eliminates its mechanical effects on the walls of the portion of the bowel and promotes a more rapid return to normal peristaltic function. *When this occurs the tube is carried farther into the inactive intestine*, decompressing as it progresses, until such time as the entire inactive bowel is completely relieved and returns to adequate peristaltic function. Much the same process produces decompression of a mechanically obstructed bowel. (2) The second group of cases include various types of mechanical obstruction and the treatment includes preoperative intubation and operation. This is the group of cases which are very often mismanaged. While realizing the value of preoperative decompression, great caution should be observed not to permit a simple mechanical obstruction of the small intestine to become converted into the gangrenous type. Wangensteen places a great deal of significance of abdominal tenderness associated with intestinal colic as a diagnositic sign of strangulation obstruction. A variable amount of abdominal rigidity is also present, however it is not a reliable sign. Because of the nature of the anatomy of the small intestine, the danger of gangrenous changes is ever present and one is never certain as to when this will occur. *For this* reason, we do not believe that surgery should be postponed until the bowel is completely deflated, nor wait for signs of gangrene. The mortality rate in gangrenous obstruction is entirely too high and its reduction is possible only when surgery is instituted at an early stage. (3) The third ground of cases are those which are indubitably gangrenous and the treatment of choice is early operation followed by postoperative intubation. In this group are included all cases falling under the classification of gangrenous obstruction as previously discussed.

OPERATIVE MANAGEMENT

Sample Obstruction.—The foregoing proceedures will correct the effects of obstruction, however early relief of obstruction or removal of its cause is of utmost importance. *No hard and fast rule can be laid down as to the choice of operation.* Each case must be studied individually, and many factors must be taken into consideration.

In simple high obstruction in the pyloro-duodenal region, a short circuiting operation, such as gastrojejunostomy, will suffice. If the obstruction is caused by

spasm of the pylorus, Rammsted's pyloroplasty is the operation of choice, especially if the patient is greatly debilitated, or if the patient is an infant with congenital pyloric stenosis. Acute duodenal obstruction is relieved by a duodenojejunostomy. In the less acutely ill patient conservative treatment, consisting of elevating the foot of the bed, will stop the vomiting and permits feeding of a high fat diet to increase the bulk of the mesentery. Acute dilation of the stomach following abdominal operations may be due to compression of the duodenum. Turning the

Fig. 538.—Method of reducing incarcerated intestine.

patient over on the abdomen will often relieve the condition. Routine postoperative [illegible]

operations and in operablle malignancies, resections.

Surgical management of obstruction of the small intestine depends entirely upon the findings. The most frequent cause is adhesions and the last 3 to 6 feet of the ileum is usually involved. In some cases a thick band may be the cause and its division and excision will immediately restore the peristaltic waves. Nothing else needs to be done. When numerous coils are adherent, freeing of these is essen-

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tial. If the adhesions are firm and old, it may be necessary to resect part of the bowel. In some cases neither may be possible and the only procedure left is an entero-enterostomy.

In simple obstruction of the large bowel a temporary colostomy is the operation of choice. This subject is more thoroughly discussed in the Chapter on Surgery of the Large Bowel.

Gangrenous Obstruction.—In gangrenous obstruction the absorption of toxins plays an important role in the cause of death. Obviously recovery can be expected only when the toxins are adequately and completely drained. In the severely toxic patient with obstruction of long duration, complete drainage is best obtained by utilizing the two ends of the bowel as a "double barrel" enterostomy after the gangrenous segment has been resected. In this manner the highly toxic contents are adequately drained. Primary resection and anastomosis should be avoided since the contents are not thoroughly drained, even though an enterostomy and intubation are instituted. Furthermore, the anastomosis opens new avenues for absorption of the toxins. It is also dangerous because the tissues are friable, edematous, and tear easily. In early cases, or when toxemia is at a minimum, a primary anastomosis may be performed with safety. This is especially true of strangulation obstruction in a hernial sac. They are usually recognized early and operated before grave effects of toxemia develop. In these cases the involved segment should be inspected, freed of its incarceration and returned into the abdomen to determine if it is viable. Figure 538 illustrates the author's method of emptying a hernial sac of incarcerated intestine. After the sac has been opened, a loop of bowel is sought which leads into the abdominal cavity. Then with gentle pressure applied with finger or hemostat, the bowel is pushed into the abdomen. After a few inches of the bowel has been reduced, the remaining loops will slide with ease. The involved loops should be left within the abdomen for at least five minutes. At the end of this period they are brought out and examined to see if the circulation has returned. To avoid errors of identification a ligature may be applied in an avascular area of the involved segment before it is placed into the abdominal cavity. If the intestine is viable, the color will return. When pinched, the depression made will disappear, and the peristalsis will become evident.

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duodenum may be divided into four parts: (1) superior part, (2) descending part, (3) inferior part, and (4) terminal part.

The first part of the duodenum, pars superius, measures about 2 inches in length and is the direct continuation of the pylorus. It is completely enfolded by peritoneum and is therefore rather movable. However, its mobility is held in check by the hepaticoduodenal ligament. Its course is backward, to the right, and slightly upward, terminating downward into the descending part. It forms the lower border of the foramen of Winslow, and is in contact with the under surface of the left lobe of the liver and the neck of the gall bladder.

The descending portion of the duodenum is from 3 to 4 inches long. It descends along the vertebral bodies to the level of the middle of the third lumbar vertebra, where it turns to the left, and becomes continuous with the third part. It is covered by peritoneum only on its anterior surface, and for this reason it is quite immobile. The common bile duct enters its wall about 3 to 4 inches from the pyloric sphincter.

The inferior part crosses the posterior abdominal wall at the level of the third lumbar vertebra, from the right to the left. Its direction is slightly upward, terminating at the left of the upper part of the third lumbar vertebra. As its termination it is crossed anteriorly by the root of the mesentery and the superior mesenteric vessels.

The terminal portion of the duodenum ascends to the top of the second lumbar where it ends in an abrupt turn forming the duodenal-jejunal flexure. The latter is prevented from slipping down by the suspensory muscle of Treitz, a musculo-areolar ligament attached to the left crus of the diaphragm (see Fig. 539).

JEJUNUM AND ILEUM

The jejunum and ileum are attached to a fold of mesentery which runs obliquely across the abdomen to the ileocecal valve. The mesentery is composed of two layers of peritoneum connecting the jejunum and ileum to the posterior body wall. That part of the mesentery attached to the posterior body wall is from 6 to 8 inches long. This part is thicker than the long free border which envelops and suspends the small intestine. This is due to the greater amount of fatty tissue and the presence of large vascular trunks. The root of the mesentery runs obliquely from the second left lumbar vertebra to the right iliac fossa. The long free border surrounding the jejunum and ileum is from 20 to 30 feet long. Because of the differences in length of the root and the long free border of the mesentery, the small intestine is thrown into numerous folds and coils.

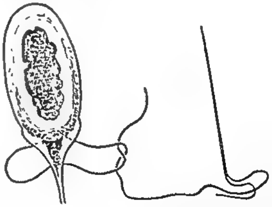


Fig 540.—Diagram to illustrate the mesenteric angle and the application of a suture to obliterate the space.

The manner by which the serosa envelops the small intestine is of surgical importance. The whole circumference is covered with the exception of the mesenteric attachment, where a triangular space is formed. This space is called the mesenteric triangle. Within this space are found lymphatic filaments, collateral blood vessels,

CHAPTER 65

SURGERY OF THE SMALL INTESTINE—ANATOMY

By A. V. Partipilo

DUODENUM

The small intestine is divided into three parts: duodenum, jejunum, and ileum. The duodenum is from 10 to 12 inches long, and extends from the pylorus to the duodeno-jejunal flexure. It varies in its course from a U to a C shape, with the concavity of its curvature being directed upward and to the left (Fig. 539). The

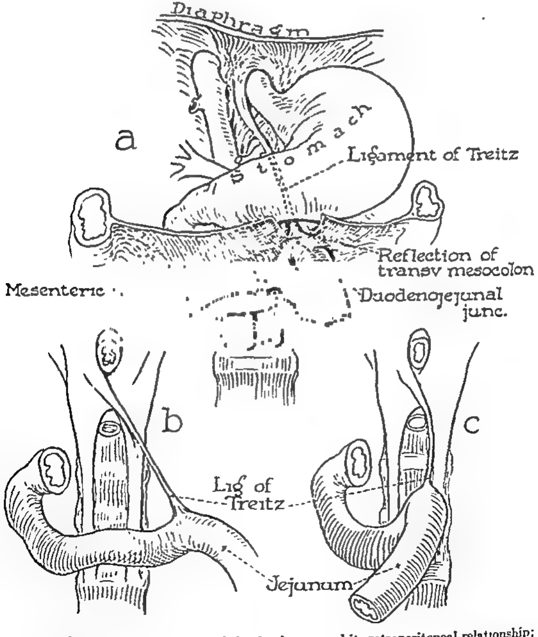


Fig. 539.—(a) Schematic presentation of the duodenum and its retroperitoneal relationship; (b) elongated ligament of Treitz, C-type of duodenum; (c) represents U-type of duodenum.

ileum, the mesentery is quite opaque and filled with fat. The mesentery at the terminal portion is rather large, and numerous tabs of fat are seen. The vessels appear as mere grooves in the fat.

FIG. 541.—Method of determining the direction of a loop of bowel; (a) the index finger and thumb are placed on either side of the mesentery; and (b) the bowel is twisted until all kinks have been eliminated.

METHOD OF DETERMINING THE DIRECTION OF A LOOP

The direction of a loop of bowel can be determined as follows: The index finger is placed on one side of the mesentery of a loop of bowel and the thumb on the other side (Fig. 541). The thumb and index finger are gently pushed downward to the root of the mesentery by eliminating all kinks and twists. When the bowel is

nerves, and fat. During an anastomosis this space must be closed in order to prevent spreading infection along the mesenteric lymph channels (See Fig. 540).

The muscularis of the small intestine consists of an outer longitudinal and an inner circular layer. Its thickness is about 4 millimeters, however, it diminishes in thickness to within two feet of the cecum, where it again becomes thicker in order to overcome the resistance offered by the ileocecal valve. The submucosa consists for the most part of connective tissue and a few elastic fibers. This layer has the strongest holding power and for this reason it must always be included in the sutures during an anastomosis. Within this layer are found blood vessels, lymphatics, and the nerve plexus of Meissner.

The mucosa, the innermost layer, is very lax, forming many folds called plicae circularis. It contains glandular elements and lymphatic nodules. The latter are of two types: (1) the solitary nodules found in the jejunum; and (2) Peyer's patches, which are collections of solitary nodules found near the lower portion of the ileum.

INTESTINAL LOCALIZATION

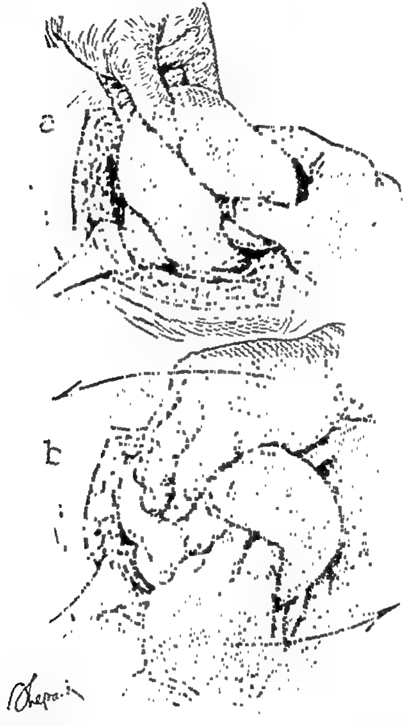
A valuable aid to determine the distance of a loop of bowel from the ligament of Treitz is the method described by George H. Monks. His method is based upon the anatomical differences of the various loops of the small intestine.

To localize a loop of intestine which may present itself through an abdominal incision, it is necessary to know which loop is most likely to present itself, and to know *the general characteristics of the various segments of the small intestine.* As a rule, the uppermost third of the intestine occupies the large cavity on the left side of the abdomen, high up underneath the ribs; the middle third occupies the middle part of the abdomen and the iliac fossa; and the lowermost third fills the pelvis and the right iliac fossa. The surgeon, knowing the compartment through which he has made the incision, can roughly determine the loop of intestine which he is likely to encounter.

The general characteristics of the intestine in the upper part differ from those in the lower part. However, this is not abrupt, but gradual. The uppermost part *is of larger caliber and thicker. By holding the bowel between the* thumb and fingers, and gently stroking it downward, the plicae circularis (valvulae conniventes) can be distinguished. They are folds of tunica mucosa with prolongations from the submucosa. The uppermost part is also very vascular, having long, large, and straight vessels in the mesentery. The vessels radiate from the gut, and form arches which are primary. The space between the vessels, called lunettes, are extensive and translucent.

For about 6 feet the gut is of smaller caliber, the vascularity is less, and secondary loops are most prominent. The lunettes are also present, but they are smaller. The vasa recta, straight vessels, are also smaller. At 6 feet the secondary loops are large; the vasa recta are somewhat irregular and show branches. No lunettes are present. The mesentery is streaked with fat and is therefore somewhat opaque. At 12 feet the vessels are smaller and the primary loops are lost in the fat. However, the secondary loops, and even the tertiary loops are visible. The vasa recta are more irregular and have numerous branches. The amount of fat is correspondingly increasing and the diameter of the bowel is becoming smaller. At about 17 feet the mesentery is entirely opaque and small tabs of fat begin to appear along the mesenteric border. The vessels are now seen with difficulty. At 20 feet, the last of the

ileum, the mesentery is quite opaque and filled with fat. The mesentery at the terminal portion is rather large, and numerous tabs of fat are seen. The vessels appear as mere grooves in the fat.



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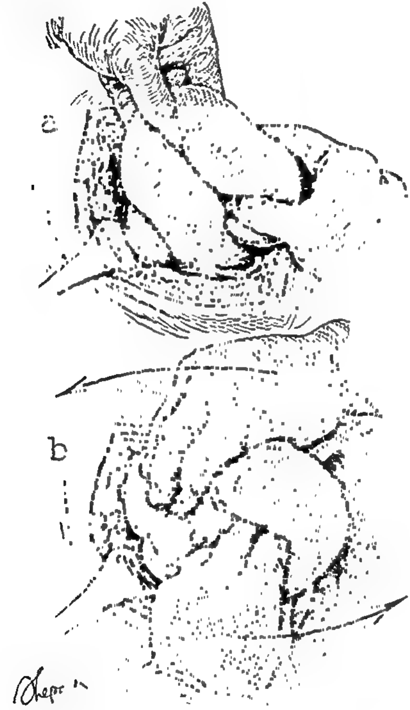

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It will be recalled that when the bowel is cut transversely, the mesenteric border is not covered with peritoneum (Fig. 544). A complication that may result from imperfect peritonization is delayed healing, with the possibility of leakage The space is theoretically vulnerable for the spread of infection through the exposed lymphatics. To avoid this possibility the exposed surfaces can be approximated at a distance from each other (Fig. 545).

Another disadvantage of an end-to-end anastomosis is the insufficient blood supply to the cut edges of the bowel, especially to that part opposite the mesenteric attachment. The blood vessels of the small intestine are practically end arteries, *i.e.*, there is no free communication of the arteries of one side with those on the other. Furthermore, the arteries are almost straight tubes. For this reason, when the bowel is cut transversely, the edges do not have a free and adequate blood supply. When two such ends are anastomosed, there is always the possibility of sloughing

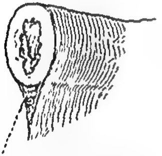

FIG. 544.

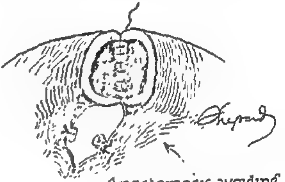

FIG. 545.

FIG. 544.—Cross section to illustrate the absence of peritoneum at the mesenteric attachment.
FIG. 545.—Method of approximating two ends of bowels in order to avoid approximating two non-peritonized surfaces.

especially at the a-mesenteric border. As illustrated in Figure 549 a, when the excision is made straight across the bowel the blood supply is poor, whereas when the incision is made at oblique angle the blood supply is increased. Of course, the obliquity of the incision must always be made in the direction away from the pathological segment. If the bowel is cut with the obliquity toward the pathology, the bowel extending beyond the a-mesenteric point will be completely devoid of blood supply. In the latter case, the inevitable result will be gangrene followed by sloughing.

Another objection to an end-to-end anastomosis is the possibility of narrowing of the lumen by inversion of the septum, or secondary obstruction, as a result of cicatricial contraction of the anastomotic ring. Fortunately this is not a common complication.

TECHNIQUE OF LATERAL ANASTOMOSIS

Resection of the Bowel.—When resecting a segment of the small intestine the first consideration is to determine the extent of the pathology and the amount of bowel to resect. The line of excision must be in good healthy bowel to insure solid and firm suturing. It is far better to sacrifice 2 or more inches of healthy bowel than to anastomose devitalized bowel. When the sites of section have been determined, mesenteric stitches are placed at the two limits of excision (see Figure 546). When

stitches are inserted from the outside, as when applying Cushing sutures, and left loose. Place a finger over the opened angle and invaginate it by applying pressure, at the same time pull the suture (Fig. 566 *a, b, c*). After the inner row is completed the clamps are removed, the operating field is cleansed, packs are removed, and gloves are either washed or changed. The anastomosis is examined for puckering

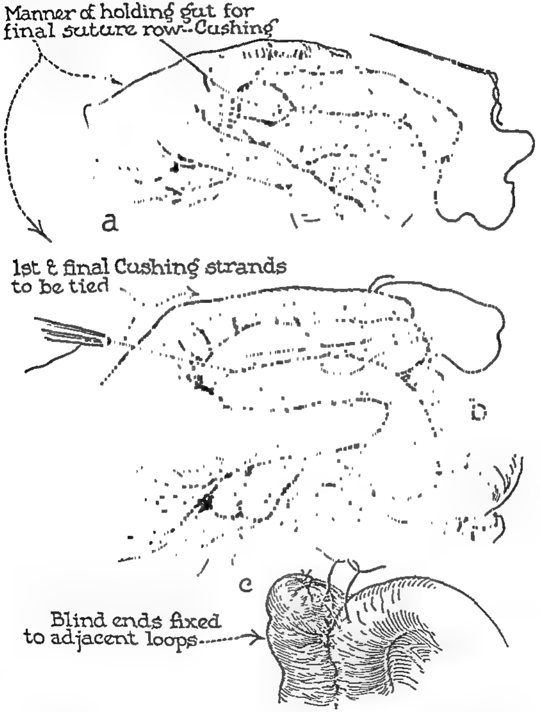

FIG. 568.—Method of holding the bowel while inserting the anterior outer row of Cushing stitches.

and for bleeding points; if present, apply a mattress suture and tie it over a forcep as illustrated in Figure 567.

The anterior outer row of Cushing stitches is now applied using the original suture that was saved after the completion of the posterior outer row. To facilitate suturing, the anastomosis is held with the middle finger underneath the posterior

surface (Fig. 568 *a*), and the index finger and thumb are placed anteriorly on either side of the suture line. In this manner the anastomosis is grasped firmly and the Cushing stitches are applied without difficulty and with greater accuracy. When completed, it is tied to the short end of the beginning of the *posterior row*.

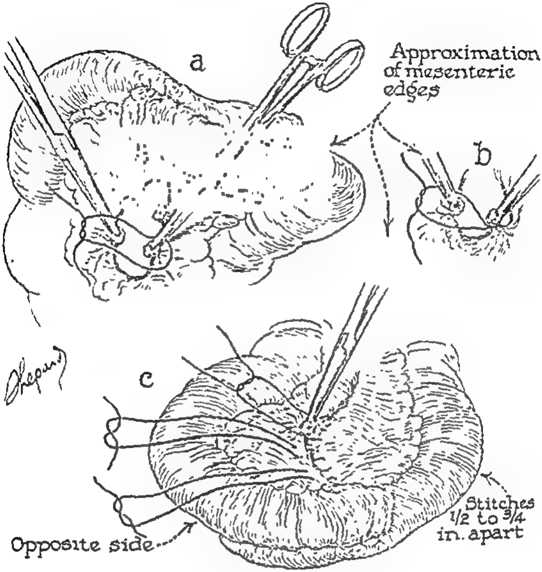

FIG. 569.—Approximation of the severed mesenteric edges; (*a*, *b*) forceps method; or by (*c*) interrupted mattress sutures.

After final inspection, the blind pouches are secured to the side of the bowel with one or two interrupted mattress stitches. The mesenteric gap is closed with interrupted or continuous sutures, or the mesentery may be grasped with forceps, then tied with catgut (Fig. 569). After final inspection the abdomen is closed without drainage.

CHAPTER 67

SURGERY OF THE SMALL INTESTINE

END-TO-END ANASTOMOSIS

By A. V. Partipilo

An end-to-end anastomosis is preferred by many surgeons because it is a physiological operation. It has certain disadvantages however, which must be taken into consideration. An end-to-end anastomosis requires diligent attention to the minutest detail, hence when performed by the general surgeon it is not the safest procedure. There is also the danger of immediate stricture as a result of inversion of the septum, or cicatricial contraction after healing has occurred. Because of the absence of peritoneal covering the mesenteric angles when sutured together are weakest part of the anastomosis. For this reason leakage and infection may occur. Undoubtedly an end-to-end anastomosis is the preferable operation when attention is given to the fine technical details to avoid the dangers mentioned.

Technique.—The method of determining the line of resection and of shutting off the mesenteric vessels and the mesenteric triangle is the same as described under lateral anastomosis. In an end-to-end anastomosis there are two factors which must be adhered to: first, the blood supply to the edge of the bowel must be adequate; and second, the line of section must not be traumatized.

After the blood supply has been cut off and the mesentery of the segment of the bowel has been incised, rubber protected flexible clamps are applied to control the contents. These are applied about 1½ inches below the lines of incision. Ordinary forceps are applied to control the contents of the segment to be removed. These are placed sufficiently away from the lines of incision to avoid traumatizing the edges of the bowel to be anastomosed.

Using scissors, the intestine is severed just within the mesenteric stitches. To insure good blood supply at the a-mesenteric border the incision is made with an oblique angle away from the pathology (Fig. 570). Scissors are preferred because they produce a clean-cut surface. To avoid ragged and traumatized edges make the cut with one snip of the scissors. The opened ends of the intestine are now cleansed with gauze and the peritoneal cavity is walled off with warm saline packs.

Before the suturing is begun, th[illegible]
the bowel. [illegible]
stitches. The two intestinal clamps are now brought together and held by grasping the rubber tubings with forceps.

The suturing is started by applying a U-stitch as the mesenteric angles. The purpose of this stitch is to close the mesenteric triangle; hence if the mesenteric stitches have been applied already, the U-stitch is not necessary. This suture includes all the angles of the two opened edges of the bowel, and for this reason there is danger of carrying an infection into the mesenteric spaces. It also adds to the amount of suture material to be buried. To avoid this, the suturing may be started with a through-and-through stitch avoiding the mesenteric spaces. This is tied, cut short, and a forceps placed on the short end (Fig. 571). The assistant holds this while the surgeon sutures the septum.

surface (Fig. 568 *a*), and the index finger and thumb are placed anteriorly on either side of the suture line. In this manner the anastomosis is grasped firmly and the Cushing stitches are applied without difficulty and with greater accuracy. When completed, it is tied to the short end of the beginning of the posterior row.

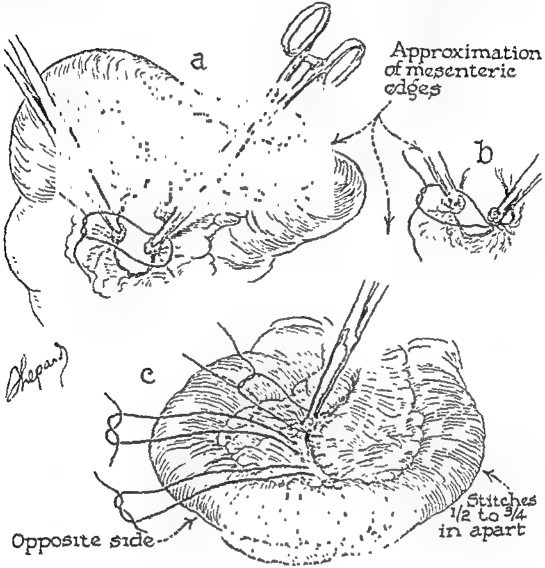

FIG. 569.—Approximation of the severed mesenteric edges; (*a*, *b*) forceps method, or by (*c*) interrupted mattress sutures.

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Using scissors, the intestine is severed just within the mesenteric stitches. To insure good blood supply at the a-mesenteric border the incision is made with an oblique angle away from the pathology (Fig. 570). Scissors are preferred because they produce a clean-cut surface. To avoid ragged and traumatized edges make the cut with one snip of the scissors. The opened ends of the intestine are now cleansed with gauze and the peritoneal cavity is walled off with warm saline packs.

Before the suturing is begun, the mesentery is scraped for a short distance from the bowel. This is done with short snips of the scissors; avoid cutting the mesenteric stitches. The two intestinal clamps are now brought together and held by grasping the rubber tubings with forceps.

The suturing is started by applying a U-stitch as the mesenteric angles. The purpose of this stitch is to close the mesenteric triangle; hence if the mesenteric stitches have been applied already, the U-stitch is not necessary. This suture includes all the angles of the two opened edges of the bowel, and for this reason there is danger of carrying an infection into the mesenteric spaces. It also adds to the amount of suture material to be buried. To avoid this, the suturing may be started with a through-and-through stitch avoiding the mesenteric spaces. This is tied, cut short, and a forceps placed on the short end (Fig. 571). The assistant holds this while the surgeon sutures the septum.

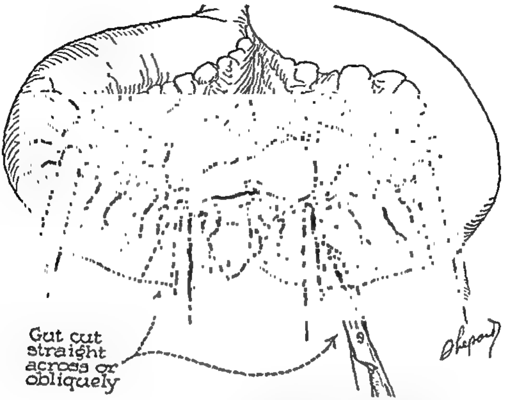
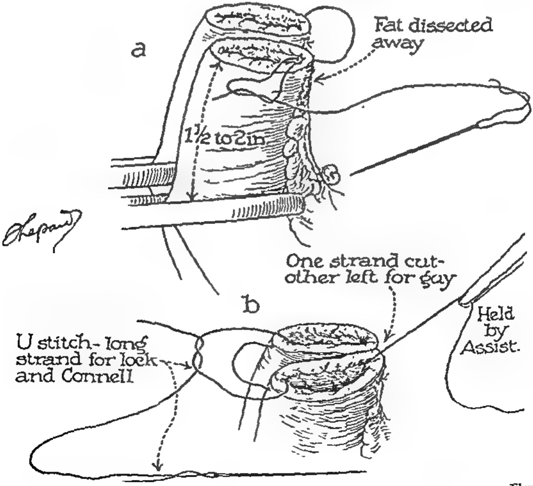


FIG 570.—End-to-end anastomosis Bowel is cut at an oblique angle to insure adequate blood supply. Mesentery has been separated and blood vessels ligated.



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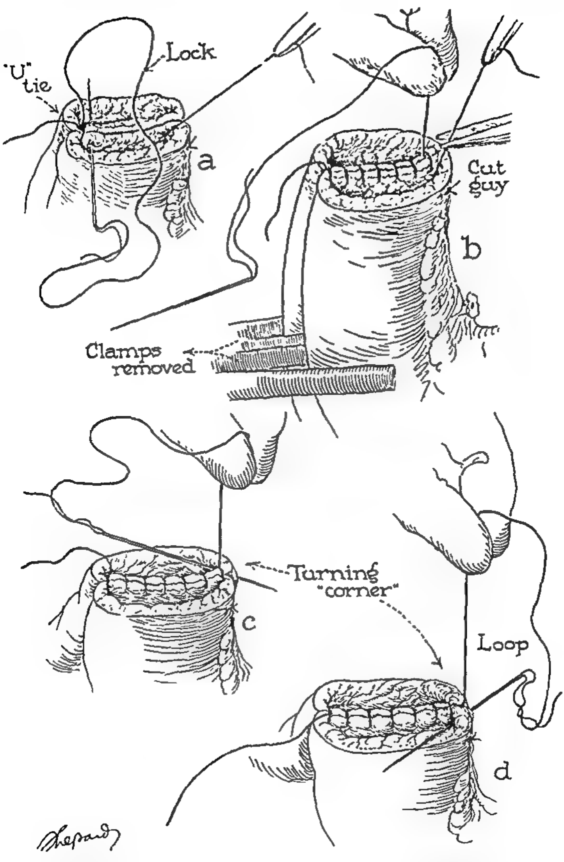

FIG. 572.—End-to-end anastomosis; (*a*) U-stitch tied and septum is sutured with a continuous lock stitch. The initial mattress is cut; (*c*, *d*) the corner is turned.

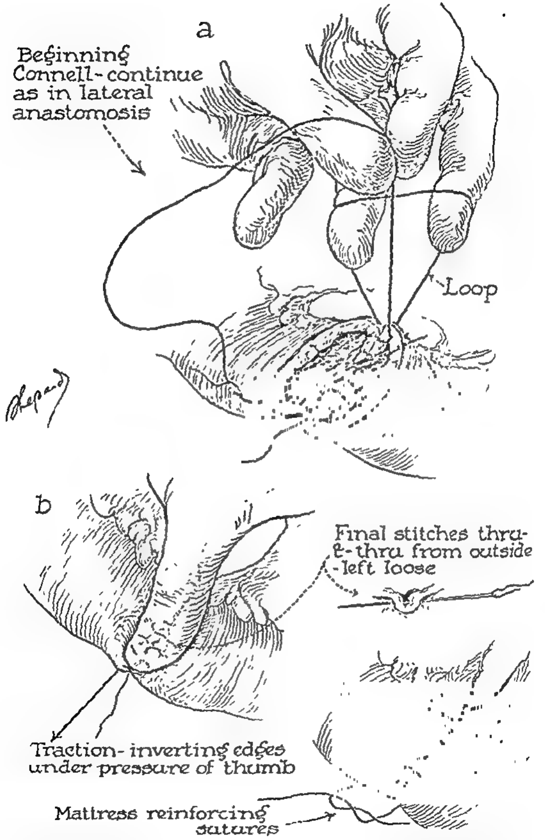

FIG. 573.—End-to-end anastomosis, (a) inversion of the anterior edges with a Connell suture, (b) terminal Connell sutures to invert the angle, (c) interrupted mattress sutures for reinforcement.

Beginning at a point opposite the preliminary stitch, the septum is sutured with an over-and-over, and through-and-through suture. While suturing the assistant holds taut the preliminary stitch, while the surgeon holds the needle end. In this manner the septum is held steady, thus making it possible to introduce the needle with greater ease. It is important to see that the peritoneal layer is included in each bite. The stitches are applied $\frac{1}{8}$ inch apart and $\frac{1}{8}$ inch deep, and may be self-locking or simple-continuous sutures without locking. The author prefers locking because the edges are approximated with greater security and the locking assures hemostasis.

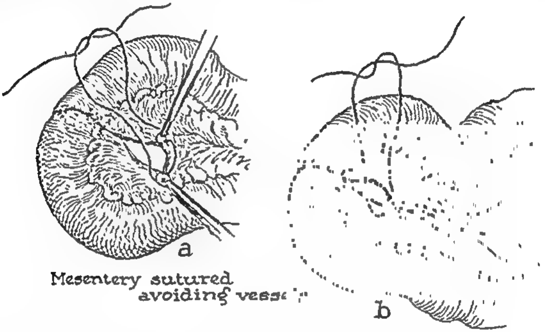

FIG. 574.—End-to-end anastomosis. Alternate methods of closing the mesenteric gap.

When the septum is entirely sutured, the preliminary stitch is cut and the corner is turned with an out-and-in suture. After inverting the corner the suture is continued as a Connell. When the anterior edges have been completely inverted the suture is tied and the ends cut short.

After the inner row of suture is completed the surgeon can decide whether a second row is necessary, or whether interrupted sutures will suffice for reinforcement. One row of sutures has been found adequate to hold the anastomosis together until healing takes place. Two rows of sutures are undesirable as the second row will increase the amount of inverted tissue, thus augmenting the danger from obstruction.

The anastomosis is now examined for bleeding points and puckering, and if found, they are taken care of with mattress sutures as described under lateral anastomosis. The mesenteric gap is closed as illustrated in Figure 574.

CHAPTER 68

SURGERY OF THE SMALL INTESTINE—END-END-TO-SIDE ANASTOMOSIS

By A. V. Partipilo

This operation* combines the technical principles of both the lateral and end-to-end anastomosis. When performing and end-to-end anastomosis there is always the danger of primary stenosis if too much tissue is inverted, or secondary stenosis from cicatricial contraction. Again, two rows of suture cannot be used without sacrificing space, thereby adding to the possibility of obstruction. These

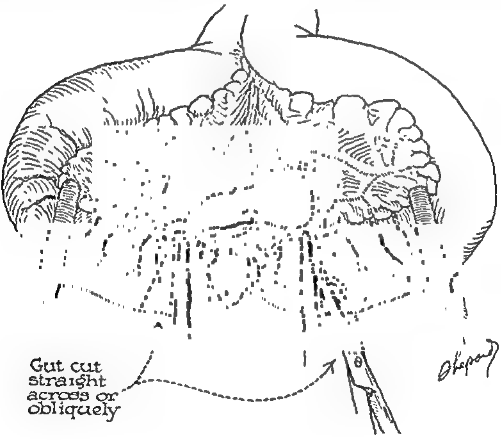

Fig. 575.—End-to-end-to-side anastomosis. Resection of the gangrenous segment.

adverse features are eliminated by an end-to-end-to-side anastomosis. It has the advantage over the lateral anastomosis in that it takes less time to perform the operation. It has distinct advantage when bowel lumina of different calibers are to be anastomosed.

Technique.—The method of ligating the central and collateral blood vessels and the excision of the gangrenous bowel is the same as described in the chapter on

* Partipilo, A. V.: End-to-end-to-side Anastomosis, Am. J. Surg., Oct. 1928.

lateral anastomosis. After the bowel is resected the two ends are approximated and a traction stitch is inserted at the mesenteric border and another is applied at the a-mesenteric border (see Figure 576). These are held taut and an incision is made along the a-mesenteric border as illustrated in Figure 577. The length of this incision will depend upon the diameter of the bowel lumen. If the anastomosis is being done upon the jejunum the incision may be made as long as the diameter of its lumen, while if being done on the ileum the incision may be made longer. When anastomosing ends with different calibers, the a-mesenteric incision upon the bowel with the smaller caliber is made so that the circumference of the two ends are equalized.

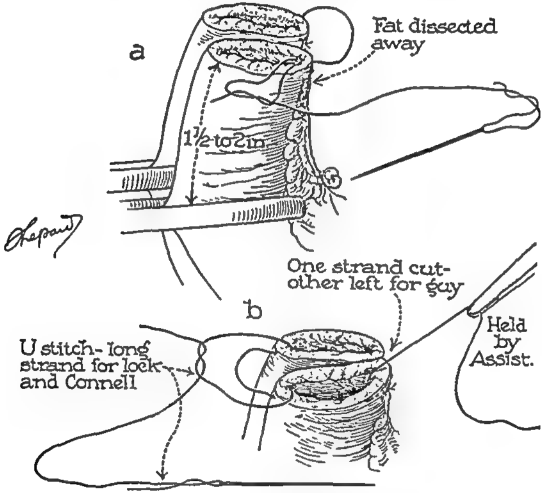

FIG. 576.—End-end-to-side anastomosis; (*a*) mattress suture at the mesenteric angle; (*b*) U-stitch at the a-mesenteric border.

After the desired length of the incisions at the a-mesenteric borders have been made, another traction stitch is taken at a point where the ends of the incisions meet. In order to avoid a valvular obstruction, the sharply angulated septum is cut with scissors (Fig. 577 *b*) and another guy stitch is taken to approximate the tissues (Fig. 578 *a*). With the assistant holding this taut the operator sutures the septum, and when the first traction stitch is reached, it is cut. The assistant then holds taut the preliminary traction stitch at the mesenteric border. When the entire septum has been sutured the guy suture is cut and the corner is inverted as described in the Chapter on End-to-End Anastomosis, p. 775. The anterior edges are inverted by a Connell suture.

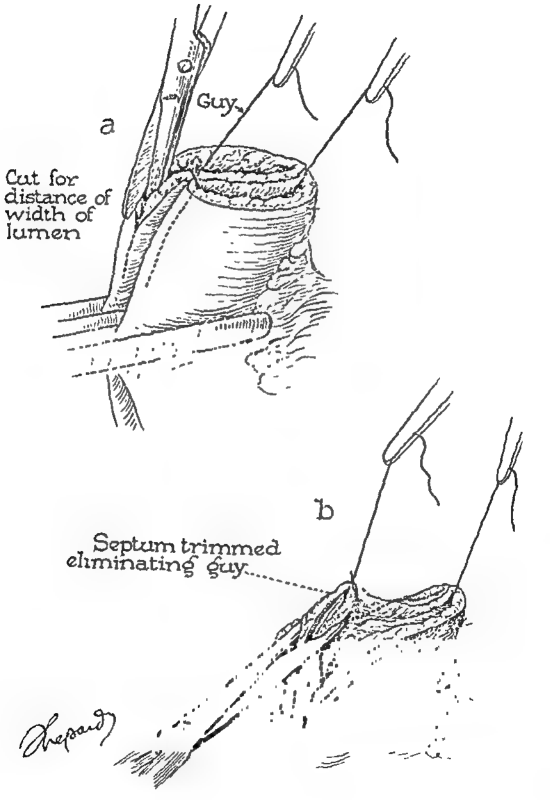

FIG. 577.—End-end-to-side anastomosis, (a) incision along the a-mesenteric border to increase the circumference, (b) septum trimmed away to avoid the formation of a valve.

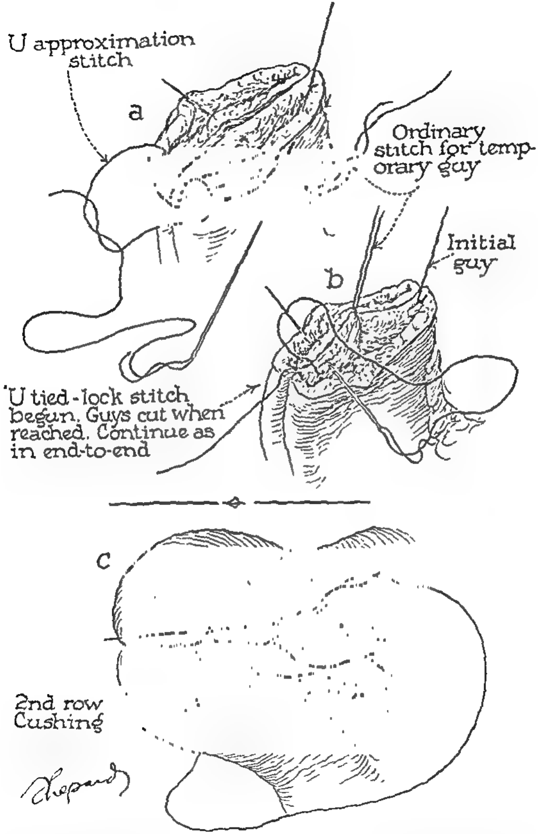

FIG 578.—End-end-to-side anastomosis; (a) stitch as a temporary guy; (b) suturing of the septum; (c) the anterior edges inverted and an outer row of Cushing sutures applied.

With this modification of an end-to-end anastomosis, a second row of Cushing may be applied without danger of turning in too much tissue. Even with two rows of suture the lumen will be equal to the original opening of the bowel. The anastomosis is a true end-to-end and a side-to-side anastomosis. It has a slight angulation which disappears in time. This operation is more physiological than a lateral and is stronger and safer than an end-to-end anastomosis.

Figure 570 illustrates the technical details of using the Murphy button when performing an end-to-end anastomosis. The button is of historical value only.

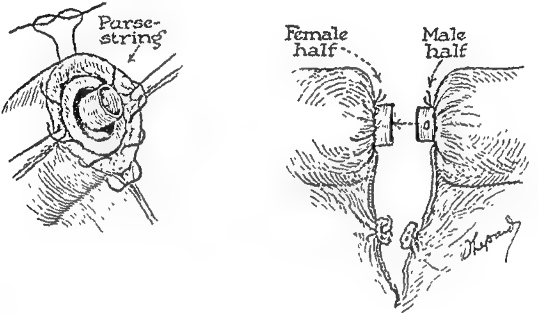

Fig. 570.—Murphy button. Purse-string suture is inserted and tied as illustrated. The two halves of the button are approximated and pressed together. The male half (heavier) is placed into the distal segment.

CHAPTER 69

SURGERY OF THE SMALL INTESTINE—ENTEROSTOMY

By A. V. Partipilo

Definition.—The term enterostomy designates the formation of an opening or a fistula in the small intestine which communicates with the outside of the abdominal wall. The terms jejunostomy and ileostomy refer respectively to openings made in the jejunum and ileum. Enterostomy is also designated as high or low; a high enterostomy is one made close to the duodeno-jejunal flexure; a low enterostomy is one made in the ileum.

Indications.—An enterostomy is a temporary measure, now infrequently employed for the purpose of draining the proximal loop of an obstructed bowel. It is now chiefly used in chronic ulcerative colitis and as a preliminary step in colectomy for polyposis, and as a complementary procedure in large bowel resections. The Miller-Abbott tube has largely replaced enterostomy as a means of draining and deflating the small intestine.

Jejunostomy is occassionally done as a means of introducing fluids and nourishment in patients suffering from advanced carcinoma of the pylorus, where gastrojejunostomy is an impossible procedure. It is also indicated in cicatricial contraction of the stomach from swallowing caustics.

METHODS

Witzel's Enterostomy.—This method was used before the Miller-Abbott tube was advocated, to lessen or prevent pressure by gas on the suture line, and subsequent leakage and peritonitis. It does not divert the fecal stream and thus is not a substitute for single-barrel ileostomy or cecostomy in surgical conditions of the colon. The loop of bowel selected depends upon the purpose; if it is to provide a safety valve for an ileocolostomy it is placed in the ileum 15 to 20 centimeters proximal to the anastomosis.

In this method a No. 10 or 12 soft rubber catheter is introduced through a small opening in the intestine (Fig. 580 *a*, *b*). A Lembert stitch anchors the tube, and the margin of the bowel is then inverted around the tube with a continuous Lembert suture as illustrated in Figure 580 *c*, *d*, *e*, *f*, *g*.

The tube is placed upon the bowel for a distance of one and one-half inches and a "gutter" is made for it by raising a fold on each side and suturing the folds over the tube with interrupted Lembert stitches (Fig. 581).

The operation is completed by carrying the catheter through an opening in the greater omentum, thus securing additional means of preventing leakage. As a further safeguard the line of suture of the enterostomy may be sutured to the abdominal wall.

In this method a "safety valve" is formed which efficiently prevents leakage even though the tube should be removed. Because the gutter is entirely lined with epithelial tissue, the walls adhere together when the tube is withdrawn (Fig. 581 *j*).

Mayo-Robson Jejunostomy.—Mayo-Robson-Jejunostomy is indicated when it is impossible to feed a patient through the normal channel. A loop of jejunum is knuckled and a lateral anastomosis is made about 3 inches below the knuckled bowel. An opening is then made in the bend of the loop and a catheter is inserted in the distal segment. The edges of the bowel around the catheter are inverted with Lembert stitches. The bowel is then sutured to the peritoneum and fascia (Fig. 582).

Maydl's Jejunostomy.—In this operation the jejunum is sectioned transversely 3 to 6 inches from the ligament of Treitz. The proximal end is anastomosed to the side of the distal loop at about 3 inches from the cut end, and the distal end is then

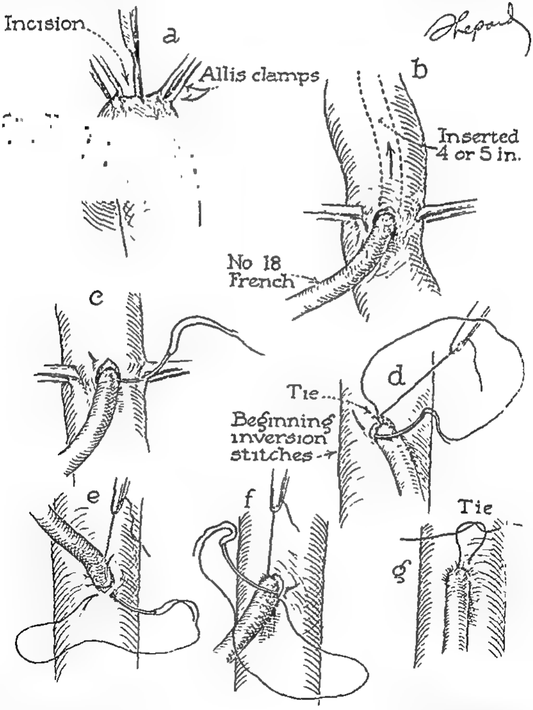

Fig 580.—Witzel's enterostomy (*a*) a small opening made in the bowel, (*b*) insertion of catheter through opening, (*c*) catheter anchored with a Lambert stitch, (*d*) the bowel opening is inverted around the catheter with a continuous Lembert suture, (*e*) another Lembert stitch anchors the tube at the half-way point, (*f*, *g*) completion of the inversion.

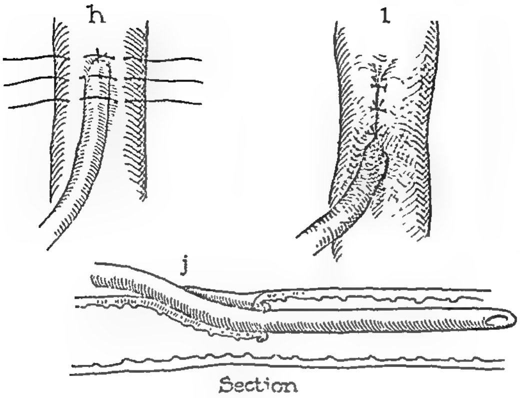

Fig. 581.—(h, i) The tube is buried within the folds of the bowel with interrupted Lembert stitches; (j) schematic sketch to illustrate the oblique valvular canal through which the tube passes.

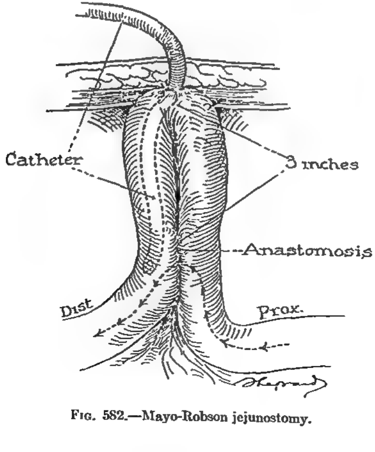

Fig. 582.—Mayo-Robson jejunostomy.

Mayo-Robson Jejunostomy.—Mayo-Robson-Jejunostomy is indicated when it is impossible to feed a patient through the normal channel. A loop of jejunum is knuckled and a lateral anastomosis is made about 3 inches below the knuckled bowel. An opening is then made in the bend of the loop and a catheter is inserted in the distal segment. The edges of the bowel around the catheter are inverted with Lembert stitches. The bowel is then sutured to the peritoneum and fascia (Fig. 582).

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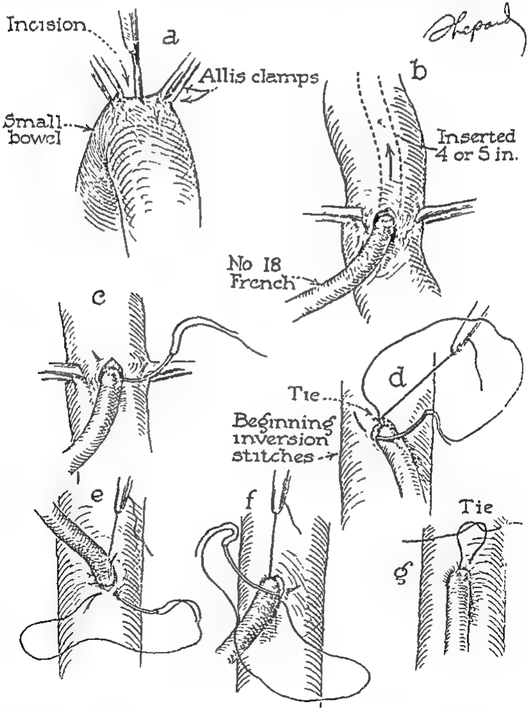


FIG. 580.—Witzel's enterostomy· (*a*) a small opening made in the bowel; (*b*) insertion of catheter through opening; (*c*) catheter anchored with a Lambert stitch; (*d*) the bowel opening is inverted around the catheter with a continuous Lembert suture, (*e*) another Lembert stitch anchors the tube at the half-way point; (*f*, *g*) completion of the inversion.

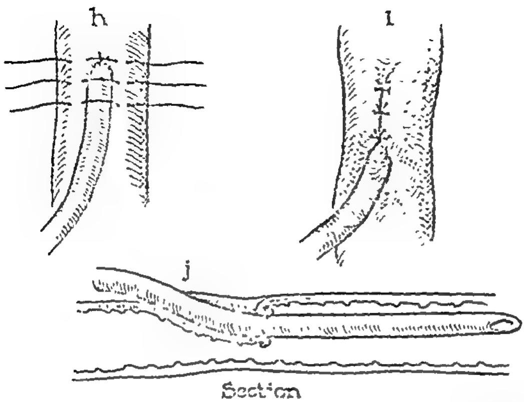


FIG. 581.—(h, i) The tube is buried within the folds of the bowel with interrupted Lembert stitches; (j) schematic sketch to illustrate the oblique valvular canal through which the tube passes.

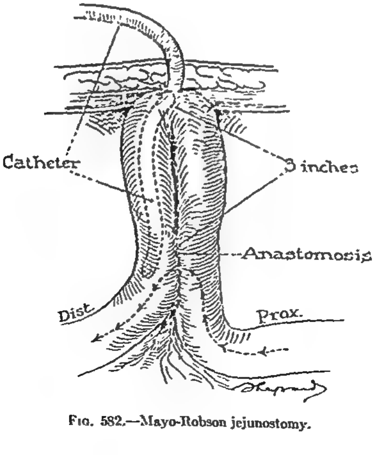


FIG. 582.—Mayo-Robson jejunostomy.

sutured to the peritoneum and skin. Thus an efficient enterostomy is formed for feeding purposes (Fig. 583).

Gun-barrel Enterostomy.—This is an emergency procedure which may be indicated in gangrenous obstruction and occasionally in a very acute case of chronic ulcerative colitis where the inflammation in the bowel and the patient's condition does not permit a more complete operation.

The technical principles of the double-barrel enterostomy are similar to ■ Mikulicz colectomy. After the diseased bowel has been resected, the distal and proximal segments are made to communicate with the outside of the abdominal wall as illustrated in Figure 584. In cases of ulcerative colitis, the ileum is knuckled together and an opening is made at the bend of the knuckled bowel without the necessity of resecting the bowel.

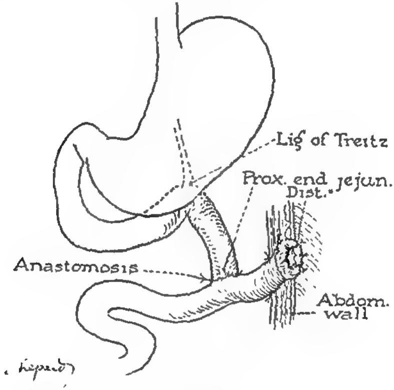


Fig. 583.—Maydl's jejunostomy.

After the patient has recovered from the effects of obstruction or colitis, the continuity of the bowel is restored according to the Mikulicz procedure (see Chapter on "Surgery of the Large Bowel," or a conventional anastomosis may be done.

A single-barrel enterostomy, as suggested by Rankin, may be done through a McBurney incision (Fig. 585). Cattell uses a right rectus incision and leaves the stoma just to the right and below the umbilicus. The technique consists of ligating a few of the mesenteric vessels of the terminal ileum and, after applying intestinal clamps to control the contents, the ileum is cut with a cautery. The distal end is inverted by the Kerr-Parker method. The proximal end ■ then brought out and sutured to the peritoneum with 5 or ■ interrupted sutures, at least 1 inch of the ileum extends above the skin. The abdominal wound is then closed in the usual manner.

Cattell brings the distal ileal limb out through a stab wound to the right, and limits the Rankin technique when a colectomy is to be done later.

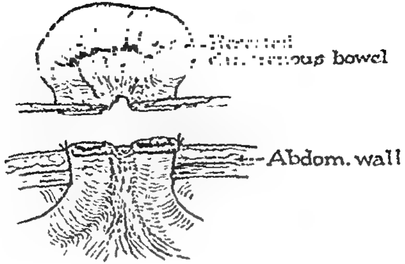


Fig. 584.—Double-barrel enterostomy.

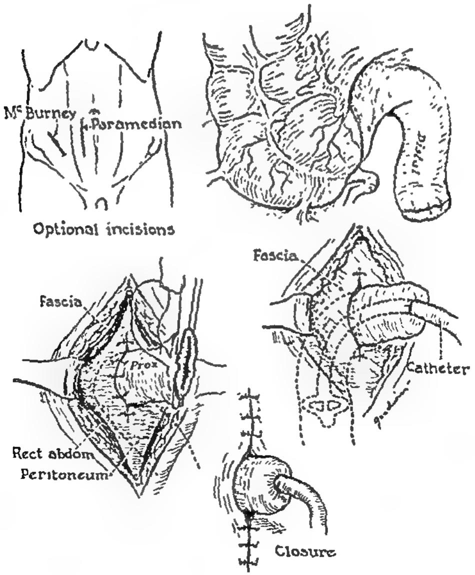


Fig. 585.—Single-barrel enterostomy (After Rankin).

Postoperative Dressings: The skin may be painted with tincture of benzoin twice a day as soon as the catheter in the ileostomy becomes loose. Later, copper, bronze, or aluminum paste applied on the skin about the stoma will usually prevent skin irritation. Kaolin or Fuller's earth has also been used. A large gutta percha circle ("washer") placed over the end of the protruding bowel and gauze dressings above this is of some value.

Skin grafting the protruding ileum is a desirable addition to ileostomy. It has been used and advocated by Dragstedt[1] since 1940. His technique follows:

Through a low right rectus incision the ileum is lifted up and divided 5 to 6 inches (13 to 15 cm.) above the cecum, the distal end closed and dropped back into the abdomen; the proximal portion is mobilized to a distance of 5 inches being careful to provide for an adequate blood supply. This is brought out through the incision with the mesenteric border inferiorly, that is at the lower angle of the wound; the stump deviates toward the mesenteric border and this makes it curve or angle downward. No sutures are placed in the bowel, one is taken in the mesentery to attach it to the abdominal wall. About 5 or 6 inches of ileum are left projecting, and the abdominal wall is closed with a few interrupted sutures and catheter is tied in the ileum.

Then a split-thickness skin graft rectangular and of proper width to cover the bowel stump is taken from the thigh or abdomen with a Padgett dermatome. This is wrapped around the bowel and sutured to the skin of the abdomen adjacent to the ileostomy wound. A snug pressure dressing is put on. About 3 inches (8 cm.) of ileum protrudes above the abdominal skin surface. A few incisions in the skin graft may be necessary to permit the escape of serum. The catheter in the ileum can be removed on the sixth to eighth day.

There is little or no excoriation of the skin when a graft is used and contractures and scarring are minimized.

REFERENCE

1. Dragstedt, L. R., Dack, G. M., Kersner, J. B., Chronic Ulcerative Colitis Ann. Surg. 114, 653 (Oct.) 1941

CHAPTER 70

ASEPTIC GASTROINTESTINAL ANASTOMOSIS

By A. V. Partipilo

Numerous methods of aseptic anastomoses have been described. A review of the literature reveals that the reason for the failure to adopt these methods is due to one or more of the following objectionable features:

1. The instruments are time-consuming and require a great deal of technical knowledge.
2. Lack of simplicity of the instrument.
3. The instrument disregards the technical principles of an aseptic anastomosis.
4. The formation of a large diaphragm which might cause primary stenosis or secondary cicatricial dimininution of the lumen.
5. Disregards primary principles of surgery.

Halsted, in 1922, described a method of colectomy which was rather ingeneous. Using experimental animals, he closed the ends of the colon and approximated them with a single row of mattress sutures. The operation was completed by puncturing the diaphragm by means of a special instrument introduced per rectum. Highsmith, in 1923, suggested a method similar to Halsted's. He closed the ends of the bowel with a strand of silkworm gut thrown around the intestines in a groove left by a narrow crushing forceps. The strand of silkworm was held in a special loop clamp, similar to a polyp snare. The closed ends were then sutured and the silkworm gut was cut by the loop clamps. The latter was removed, thus re-establishing the continuity of the lumen. Although this method was an improvement over Halsted's method, it inverted too much tissue.

Kerr and Parker, in 1923, also devised a closed method of anastomosis. They inverted the ends of the intestine over Carmalt forceps. The sutured ends were not tied, but left long. The inverted ends were then brought together and sutured with a continuous Cushing stitch. After this was done, the original inverting stitches were removed, thus re-establishing the continuity of the lumen. This method leaves a large diaphragm which may cause subsequent cicatricial contraction at the site of the stoma.

Perret of Berne, Switzerland, described a method which employed invaginators and coprostatic clamps. The essential feature of this method is the crushing of the two segments to be anastomosed, at the same time and with the same instrument. The coprostatic clamps help to hold the intestine together when the invaginators are being applied. The intestines are sutured over the invaginators with two external, continuous, circular, and concentric silk sutures. Perret used this method in a number of cases with no bad results, such as hemorrhage.

Rankin proposed a three-bladed clamp with a fixed central blade, against which the two lateral blades operate independently. When the clamp is in use the posterior peritoneal surfaces of the two arms of the bowel are in direct apposition, being separated only by the width of the central blade. After the anterior and posterior

sutures have been applied, the clamp is withdrawn and the whole line of suture is drawn taut with perfect apposition. This method gives satisfactory results. Secondary hemorrhage or the formation of a diaphragm does not occur with this method.

Horsely, in 1929, suggested a method similar to the one which the author described in the same year, except that he employed ordinary Payr. He sutured the posterior row with a continuous suture with the two blades of the Payr clamps spread apart. The clamps are then brought together, and the anterior row is sutured with interrupted mattress sutures. The clamps are then withdrawn and

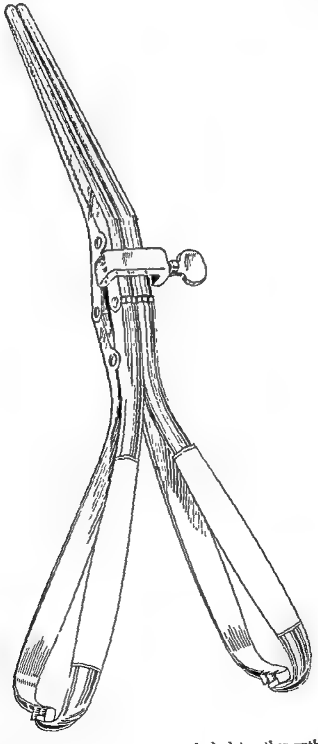
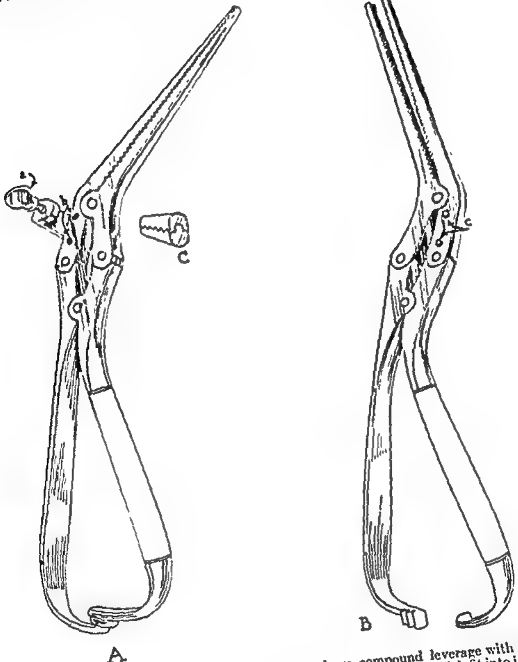


FIG. 586.—Partipilo clamp. The two clamps are locked together with set-screw. Handles are angulated to permit freedom of manipulation; also has an abdominal curvature, conforming to the curvature of the abdomen.

the mattress sutures tied. Horsley suggested this method when the colon is bound down with adhesions, or when the bowel is immobile because of its anatomical peculiarities, which makes it difficult, if not impossible, to apply the posterior row. The Payr clamps have been found rather cumbersome and not easily manipulated. The blades are also too wide, thus leaving a large diaphragm at the site of the stoma.



Clamps have compound leverage with hand grip. ... which fit into indentations ... e.

It has also been observed that when interrupted stitches are used, the agglutinated septum has the tendency to spread apart when the stitches are being tied, thus allowing the contents to escape and contaminate the peritoneal cavity. This is especially true if the bowel is distended. In cases with immobile colon the author's clamps can be used according to the suggestion made by Horsley. Since the instrument consists of two separate clamps, the posterior row of suture is applied

with the clamps spread apart. The clamps are then locked and the anterior row is applied as a continuous Cushing suture. This overcomes the objectionable feature of using interrupted sutures.

Partipilo Clamp.—In 1929, the author suggested the use of two Carmalt forceps which could be locked together by a set-screw. It was early proved that the blades were too flexible and not strong enough to crush the diaphragm thoroughly. Various forceps were tried and finally the instrument described herein was developed. The instrument consists of two compound leverage clamps having greater crushing and agglutinating power than Payr clamps (Fig. 586). When in use the two clamps are locked together by a set-screw attached to the left clamp. To prevent motion when the instrument is locked, there are two prongs on the leverage part of the right clamp which fit into indentations on the corresponding side of the left clamp (Fig. 587). In this manner, the two clamps can be locked firmly and evenly, thus permitting the surgeon to manipulate the instrument with ease while suturing. In order to facilitate opening and closing, the handles are angulated, because it is almost impossible to open two straight forceps when they are held firmly side by side. The instrument also has an abdominal curvature so that it will remain in the same position on the abdomen. The blades are angiotribed and contain cross serrations to prevent the crushed edges of the bowel from slipping. They are 5 millimeters wide and 8 centimeters in length.

TECHNIQUE V-SHAPE SECTION OF GASTRIC ULCERS

Ulcers on the Greater Curvature.—The first step is to discover the extent of the ulcer and the amount of stomach to remove. After this has been determined, guy stitches are applied at the limits of the resection on the greater curvature. The right and left gastro-epiploic vessels are included in the stitches, or if this is not possible, they are ligated separately. The gastrocolic omentum is now detached from the stomach between the two guy stitches. It is also a good plan to scrape omental fat for a distance of ½ inch from the stitches, to avoid interposition of fat (Fig. 588).

The clamps are now applied so that a V-shape piece of the stomach is included with the ulcer located at the base of the V. In this manner, after the cut edges are sutured, the transverse diameter of the stomach is not narrowed. The V-shape is defined on both the anterior and posterior surfaces of the stomach, thus actually excising a diamond piece of the stomach.

The left clamp is applied about one-eighth inch from the left guy stitch with the tip of the blade resting at a point opposite the center of the ulcer (Fig. 589). The right clamp is applied ⅛ inch from the right guy stitch, at about the same angle as the former. The two clamps outline a V, with their tips forming the apex and the greater curvature the base. Ordinary forceps are now placed to control the contents of the stomach to be resected. The stomach is incised between the clamps and the forceps with scalpel or cautery (Fig. 590). The incised stomach is discarded carefully to avoid contaminating the operating field. Phenol and alcohol are applied to the raw surfaces protruding from the clamps.

As illustrated in Figure 592, the two clamps are brought together and locked with a set-screw. The stomach is now ready for suturing. The suture material can be either silk, linen, or specially prepared gastrointestinal catgut. A double instead of a single strand is used, because if one of the strands should break, there is still one strand to hold the anastomosis.

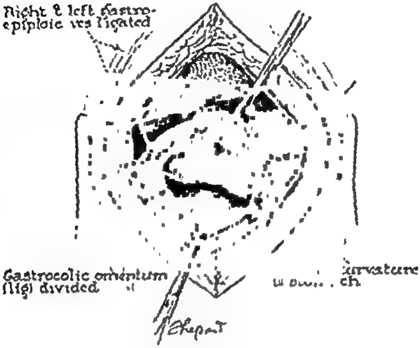


FIG. 588.—Ulcer on the greater curvature of the stomach. The gastrocolic omentum has been detached from the stomach to be resected. Guy stitches placed ½ inch on either side of the ulcer and area of infiltration.

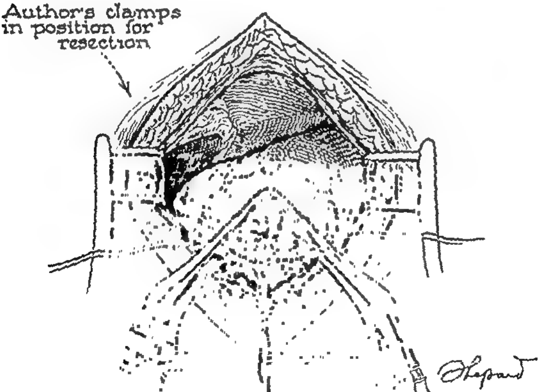


FIG. 589.—Partipilo clamp grasping the area of the stomach to be removed. They are placed one-eighth inch from the guy sutures.

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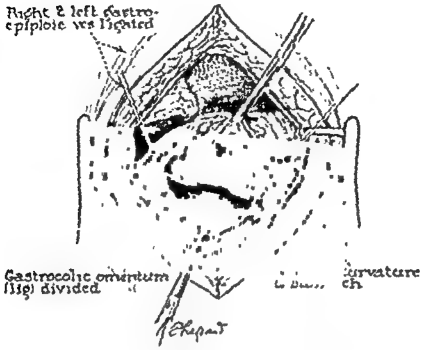


FIG. 588.—Ulcer on the greater curvature of the stomach. The gastrocolic omentum has been detached from the stomach to be resected. Guy stitches placed ½ inch on either side of the ulcer and area of infiltration.

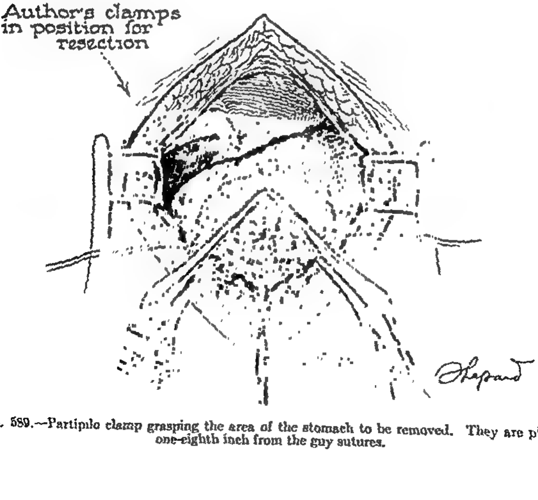


FIG. 589.—Partipilo clamp grasping the area of the stomach to be removed. They are placed one-eighth inch from the guy sutures.

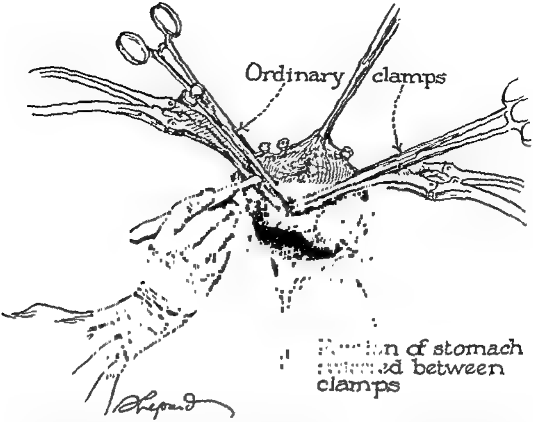


FIG. 590.—Left and right clamps applied, grasping a V-shape piece of stomach within which is found the ulcer. Ordinary forceps placed to control the contents of the stomach to be removed. *Incision made with scalpel or cautery.*

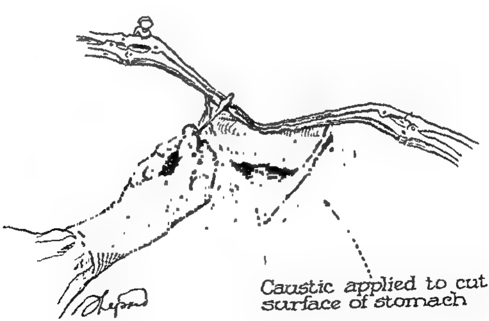


FIG. 591.—Wedge-shaped piece of stomach excised. Tissues within clamps cauterized with phenol and alcohol.

The anterior surface of the stomach is sutured first. With a straight intestinal needle a Lembert stitch is applied at the tip of the right blade. It includes all the coats, except the mucosa. The suture is continued as Cushing stitches, which are applied parallel to and ⅛ inch from the clamps, and alternating first on one side, then on the other (Fig. 593 *a*). The stitch ends on the opposite side from where it was started; *i.e.*, to the left of the left clamp at the leverage end of the instrument.

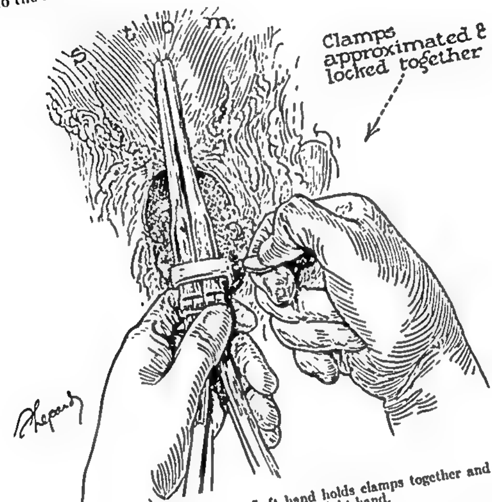
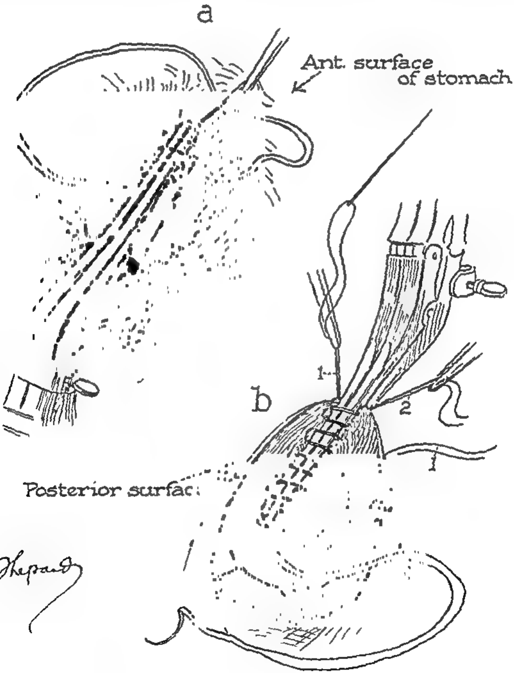


FIG. 592.—Method of locking clamps. Left hand holds clamps together and the set-screw is tightened with the right hand.

The clamp is now turned over to expose the posterior surface of the stomach. The assistant now holds the instrument, while the operator sutures the posterior surface. The suturing is the same except that a curved needle is employed. The first bite is a Lembert taken at the leverage side of the right clamp. The suture is then continued as Cushing and ends at the tip of the left clamp (Fig. 593 *b*). Thus all corners contain Lembert stitches which aid in the inversion of the corners. At the tip of the blades are found the short end of the anterior and the needle end of the posterior rows of sutures. At the leverage side are found the short end of the posterior and the needle end of the anterior rows of sutures.

After the suturing is completed, the clamps are ready to be removed. This is one of the most important maneuver in the entire procedure and requires teamwork on the part of the operator and the assistant. The assistant's duty will be to pull on the short end of the anterior and the needle end of the posterior row of sutures (Fig. 594). The needle end of the anterior and the short end of the posterior sutures

are grasped with a forceps and held by the surgeon. Notice that both ends held by the assistant are found at the tip of the instrument, while those held by the surgeon are at the greater curvature. The operator releases the blades, and with the instrument in his right hand and the suture ends in his left, begins to draw the clamps outward. As the clamps are being withdrawn the assistant draws taut the sutures which he holds. The surgeon does not pull on the suture ends at the greater curvature until the clamps are withdrawn almost entirely. The withdrawal of the instrument and the pulling of the sutures by the assistant must be done simultaneously, in order that the edges may be inverted as the clamps are being removed. After



...st stitch

stomach, shows four ends: (1) short and needle end of anterior, and (2) short and needle posterior sutures.

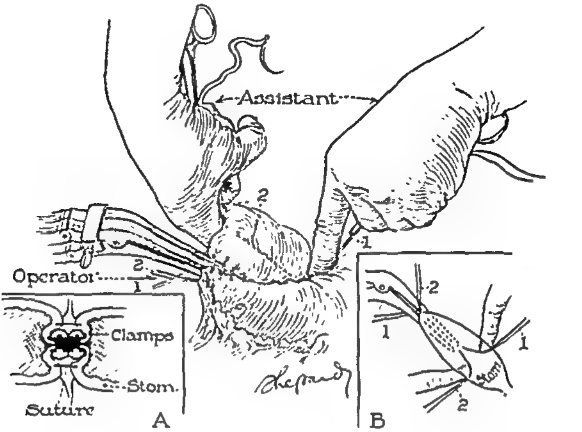


Fig. 594.—Method of removing the instrument. Blades have been released and as the surgeon [illegible] relationship of the blades and the sutures.

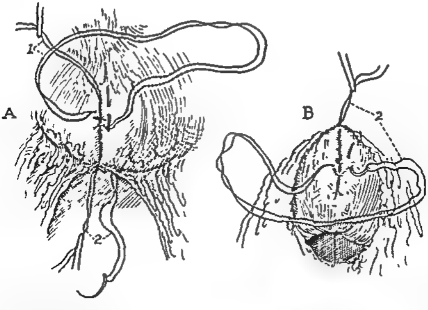


Fig. 595.—(a) Clamps have been completely withdrawn and ends tied. Second row of Cushing stitches to reinforce the inner row; (b) Placing second row of Cushing stitches on the posterior row.

the instrument is completely withdrawn, the suture line is inspected and the ends are tied. These sutures do not penetrate the entire thickness of the stomach wall, but just down to the mucosa. A second row of Cushing stitches is inserted to reinforce the inner row. Since the needles that were used for the inner row did not penetrate the mucous membrane, they may be used for the second row (Fig. 595).

Ulcers on the Anterior Surface.—Ulcers on the anterior surface of the stomach offer a somewhat different problem, although the principle of the operation is the same as that for resecting ulcers close to the greater curvature. In these cases the ulcer is removed with a diamond-shape piece of stomach. The long axis of the diamond should be parallel with the long axis of the stomach (Fig. 596). The diamond is folded to form a V, with the long axis forming the base and the point of the Partipilo clamp, when applied, the apex (Fig. 597). The ulcer should be at the base of the triangle.

Fig. 596

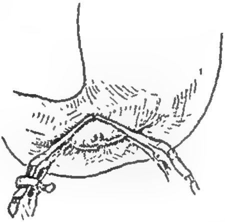


Fig. 597

Fig. 598

Fig. 596.—Ulcer on the anterior surface of stomach. Dotted line represents area to be resected
Fig. 597.—Method of applying clamps; removing a diamond-shape piece of stomach.
Fig. 598.—After completion of operation, line of suture is in the transverse diameter of the stomach.

The first step in the operative procedure is to determine the ulcer area to be resected, and to outline an imaginary diamond. With Allis forceps the stomach is grasped on the line formed by the two longest points of the diamond. By pulling on these the stomach becomes folded so that when the clamps are applied, a V-shape will be defined as illustrated in Figure 597. Forceps are then applied to control the contents of the portion of stomach which is to be removed. The latter is resected and discarded. The raw surfaces protruding from the clamps are cauterized with phenol and alcohol. The clamps are locked together, and the operation is completed in the same manner as that described for excision of greater curvature ulcers. After suturing is completed, the suture line will be in the transverse diameter of the stomach (Fig. 598), thus the diameter of the stomach is increased.

Resection of Duodenal Ulcers.—The method described above is applicable for excising ulcers of the anterior surface of the duodenum. Because of the relationship of the pancreas to the posterior surface of the duodenum, surgery of this region is difficult, if not impossible.

As in resecting ulcers on the anterior surface of the stomach, a diamond-shape piece of duodenum is folded with the long axis in the longitudinal direction of the duodenum. The clamps are applied and the suturing is done in the same manner

as described. Should the ulcer be located close to the pylorus, part or all of the sphincter may be removed with the ulcer. The advantage of this method is that the duodenum and pyloric sphincter is enlarged at the point of the anastomosis.

Ulcers on the Lesser Curvature.—The blood supply of the lesser curvature is very profuse, and for this reason it is important to ligate the right gastric vessels and the branches of the left gastric artery well away from the lines of excision. The omentum is detached from the lesser curvature between the two guy sutures. The clamps are applied, grasping a V-shape piece of the anterior and posterior surfaces of the stomach. Forceps are placed to control the contents of the stomach to be removed. This is resected, and the protruding part is cauterized. The technique in the application of the sutures and the method of pulling the instrument out is the same as for excising ulcers on the greater curvature.

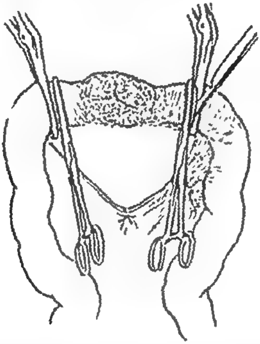


FIG. 599.—Lateral anastomosis. Partipilo clamps applied beyond gangrenous area; Carmalt forceps can be used for this purpose; diseased bowel is incised and discarded.

Lateral Anastomosis.—The first proceedure is to determine the extent of the pathology and the amount of bowel to resect. Place guy stitches at the limits of the resection. These should be taken at the mesenteric border and should include the muscular coats of the intestine. When tied they obliterate the mesenteric angle, and also shut off the collateral circulation. In this manner a bloodless operation can be performed with minimum danger of spreading infection through the lymphatics of the mesentery. The vessels supplying the gangrenous bowel are ligated separately and mesentery between the two guy sutures is divided. Forceps with longitudinal serrations (Carmalt) are applied at least ½ inch from the mesenteric stitches, and ordinary forceps are applied to control the contents of the bowel to be removed (Fig. 599). The diseased bowel is excised between the forceps, and the edges protruding out of the Carmalt forceps are carbolized, after which the ends of the bowel are inverted by the Kerr-Parker method as shown in Figure 600.

After the ends have been inverted, the two loops of intestine are placed side by side and the Partipilo clamp is applied. The left blade is applied with its tip pointing away from the blind pouch of one of the loops, whereas the tip of the right blade

points toward the blind pouch of the other segment (Fig. 601). The amount of bowel to grasp with the instrument will depend upon the size of lumen desired. Grasping $2\frac{1}{2}$ inches will give an opening about 2 inches long. Sufficient tissue should protrude from the clamps so that when it is shaved off an opening will be made into each of the segments within the clamp (Figure 601).

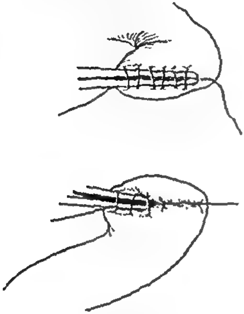


FIG. 600.

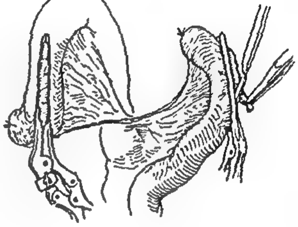


FIG. 601.

FIG. 600.—The two ends of the bowel are inverted by Kerr-Parker method.

FIG. 601.—Partipilo clamps grasping the a-mesenteric border of the two loops. Sufficient tissue protrudes out of clamps so that when they are excised an opening will be made into each segment.

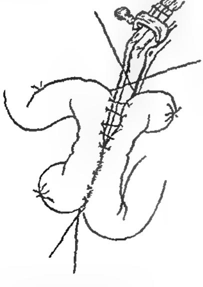


FIG 602

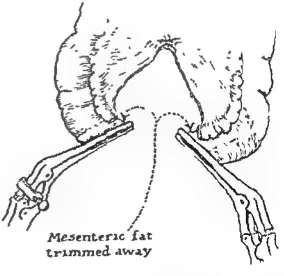


FIG 603.

FIG. 602.—Both anterior and posterior sutures have been applied. Blades are then released. Operator withdraws the clamps simultaneously with pulling of short end of anterior and needle end of posterior sutures by the assistant.

FIG. 603.—End-to-end anastomosis. Clamps applied with tips beyond the mesenteric edges. Mesenteric fat trimmed away for a short distance.

After the clamps have been brought together and locked, the same type of suture as described for resecting ulcers is used, and the operation is completed in the same manner, except that after both surfaces have been sutured the needle end of the posterior suture is delivered through the mesenteric gap. The assistant will pull on this, and the short end of the anterior row. In order to aid in the inversion of the corners, these sutures are pulled in a cross direction, which brings the edges of the bowel together and avoids pulling them apart (Fig. 602).

With the employment of the Partipilo clamp a lateral anastomosis can be performed within eight minutes. Another advantage of this method is that the blind pouches can be made rather small by applying the tips of the instrument as close to the inverted ends as desired.

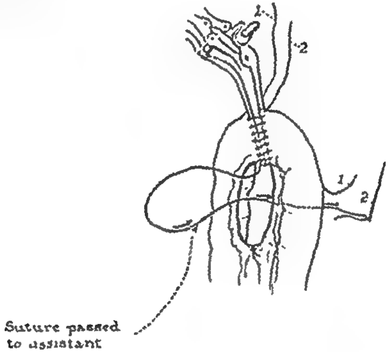


FIG. 601.—Anterior and posterior surfaces have been sutured. Needle end (2), delivered through mesenteric gap for the assistant to hold.

End to End Anastomosis.—The method of ligating the collateral and central vessels and the shutting off of the mesenteric triangle is described in the chapter on "End-to-End Anastomosis,".

A very important step in this operation is to free the mesentery from its attachment for a short distance from the line of resection. The purpose of this is to prevent the inversion of mesenteric fat within the anastomosis. Fat prevents the formation of fibrin which is an essential element in the process of healing. Inclusion of fat delays union, and the author believes that it is one of the most frequent causes of postoperative leakage after an end-to-end anastomosis.

Figure 603 shows the fat trimmed from the bowel surface close to the lines of excision. It also illustrates the application of the Partipilo clamp. To safeguard the blood supply to the edges, expecially to that part opposite the mesenteric attachment, the clamps are applied in an oblique angle. The obliquity must be made away from the diseased bowel. When applying the clamps see that the tips of the blades do not extend farther than just beyond the edges of the bowel. Should the blades extend far beyond the edges, the agglutinated septum may become separated

when the clamps are being removed, thus defeating the purpose of an aseptic anastomosis.

After the gangrenous gut has been resected the clamps are brought together and locked. The bowel is then sutured as described for lateral anastomosis. Two rows of sutures should not be employed for an end-to-end anastomosis of the small intestine because of the danger of narrowing the lumen. However, interrupted mattress sutures may be applied with safety.

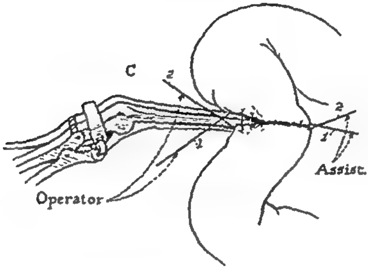


FIG. 605.—The blades have been released The assistant pulls on sutures (1, 2) in a cross direction.

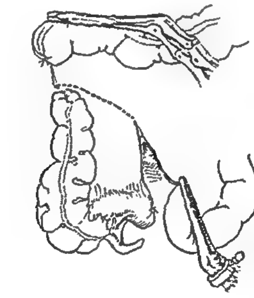


FIG. 606.—Ileocolostomy; end-to-side anastomosis after resection of cecum and ascending colon.

Figure 604 illustrates the method of delivering the needle end of the posterior row of suture through the rent in the mesentery. The assistant should pull on this and the short end of the anterior suture as the clamps are being removed (Fig. 605). The mesenteric gap is closed with interrupted or continuous stitches (see Chapter on "End-to-End Anastomosis.")

Surgery of the Colon.—The peculiarities of the blood supply, coupled with the highly septic contents of the large bowel, mitigate against successful surgery of the

colon. Undoubtedly, infection is the most serious complication. Practically every operation performed by the open method is followed by a local peritonitis which must be overcome by the patient. Closed methods abolish this factor and permit primary resections which in many cases are not possible with open methods. Nevertheless, even though performed under aseptic conditions, it must not be forgotten that successful results can be obtained only when the patient has been prepared properly for the operation, and the surgical risk evaluated. Should the risk be too great, it is best to perform a two- or three-stage operation. The Mikulicz principle of colectomy, done in two or three stages, offers the lowest mortality rate. Rankin suggested a modified Mikulicz for obstructive conditions of the colon. The operation is divided into three or four stages. During the first stage the diseased bowel is removed and the remaining bowel is mobilized by suturing the peritoneum around it. The Rankin clamp is left on the bowel to control the contents, and should the proximal loop become distended, it can be opened by releasing one side of the clamp. The bowel is prevented from retracting by leaving the other clamp closed on the other segment. If possible it is best to wait from thirty-six to seventy-two hours before opening the proximal clamp, in order to allow the peritoneum around the bowel to become agglutinated. After the Rankin clamp is entirely removed a functioning colostomy is established which is allowed to remain open for a period of two or three weeks. At the end of this period the continuity of the lumen of the bowel may be restored by cutting the septum between the two segments of the colostomy. This is done by applying forci-pressure on the spur for a period of seventy-two hours. The operation is completed by closing the colostomy in the usual manner. (For more details, see Chapter on "Colostomy.")

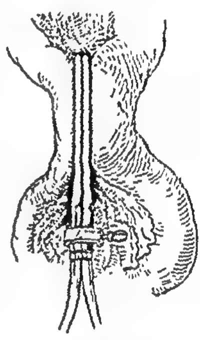


Fig. 607.—Method of applying clamps for entero-enterostomy; technique same as for lateral anastomosis.

When the condition of the patient permits a primary resection and anastomosis, a closed method of colectomy is without doubt the method of choice. Figure 606 illustrates a resection of the cecum and ascending colon with an ileocolostomy. The cecum and ascending colon are freed of their peritoneal attachment and mobilized in the usual manner. The ends of the bowel to be removed are either inverted or enclosed within rubber gloves to prevent contamination of the peritoneal cavity. A Partipilo clamp is applied on the end of the ileum, and another grasps the colon along the anterior longitudinal band. To avoid a long pouch the tip of the blade is placed close to the inverted end. The excess tissue protruding out of the clamp of the colon side is shaved off and the clamps are brought together and locked. The rest of the operation is the same as described for end-to-end or lateral anastomosis.

This procedure is also applicable for ileosigmoidostomy and ileocolostomy of the transverse colon. To a large extent the anatomical relationship of the peritoneum to the bowel wall determines the type of operation that can be performed. In the regions of the ascending and descending colon the peritoneum limits their mobility, making it impossible to perform a lateral anastomosis. In the regions of the transverse colon and the sigmoid, a colocolostomy is relatively a simple procedure.

Entero-Enterostomy.—By entero-enterstomy is meant the establishment of an opening between two loops of small intestine. This operation is indicated after anterior gastrojejunostomy, after partial gastrectomy with a long posterior loop, and for short-circuiting adherent loops of intestine.

The operative procedure consists of placing the two loops of intestine side by side and grasping about two inches of their a-mesenteric borders with the Partipilo clamp. The protruding tissue is shaved off, and the clamps are brought together and locked. The suturing is the same as for a lateral anastomosis (Fig. 607).

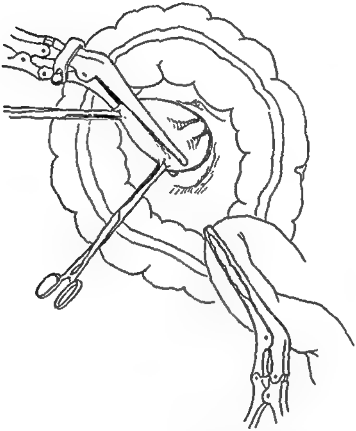


FIG. 608.—Gastro-ente[illegible] grasps posterior surface of the stomach, with tip [illegible] with [illegible] Right clamp applied on the first loop of jejunum lesse[illegible] When the two clamps are brought together the [illegible] will be approximated to the proximal end of the jejunum, and the greater curvature to the distal end.

Gastrojejunostomy.—The clamp method can be used for both the anterior and posterior gastro-enterostomy. The clamps cannot be used with facility when the stomach is adherent, nor when the mesocolon is not long enough to permit the delivery of the posterior surface of the stomach through it. Such abnormalities also contraindicate a posterior gastrojejunostomy by the open method.

The technical principles for antiperistaltic posterior gastrojejunostomy are as follows: An opening is made in the transverse mesocolon to the left of the mid-colic artery. An Allis forceps is placed through the rent of the mesocolon and the stomach is grasped close to the lesser curvature at the angula incisura. With another Allis forceps a corresponding point close to the greater curvature is grasped so that a line between the two forceps will conform to the anatomical direction of the first loop of jejunum. The stomach is then delivered through the mesocolic rent by pulling on the Allis forceps. Figure 608 illustrates the protrusion of the posterior surface of the

stomach through the mesocolon and the method of applying the Partipilo clamp. The left clamp grasps the stomach between the two Allis forceps, with its tip pointing toward the lesser curvature. Sufficient stomach should protrude so that when it is cut the mucous membrane is included. The right clamp is applied on the jejunum with its tip directed toward the proximal end of the bowel. The protruding tissue of the jejunum and stomach are now shaved, and the clamps are brought together and locked. The operation is completed the same as described for a lateral anastomosis.

For the iso-peristaltic method the incision through the mesocolon should be made transversely, when practical, to allow greater access to the posterior surface of the stomach. Two Allis forceps are applied 1 inch from the greater curvature, and 2 inches apart. To insure hemostasis, large vessels leading into the line of incision are stick-tied. The left clamp grasps the stomach, with the tip of the blade directed toward the cardiac end, whereas the right clamp is applied on the jejunum with its tip pointing towards the proximal end. After the clamps are applied and locked, the anastomosis is completed as described for a lateral anastomosis.

Gastro-Duodenostomy.—Whenever this operation is indicated, the Partipilo clamps can be used with facility. The operation is essentially a lateral anastomosis of the first and second part of the duodenum and the anterior surface of the pyloric end of the stomach.

The operative procedure consists of first making a Kocher incision to mobilize the second part of the duodenum. The duodenum and the stomach are then brought together side-to-side and the clamps are applied as for a lateral anastomosis. The right clamp is applied on the stomach with its tip toward the pyloric sphincter, whereas the left clamp grasps the duodenum with its tip also toward the sphincter. The suturing is the same as that for a lateral anastomosis.

Cholecystogastrostomy.—The author has also employed the instrument for anastomosing the gall bladder to the stomach or to the duodenum in cases of obstructive jaundice due to carcinoma of the pancreas. The operative steps consist, first, of partially evacuating the gall bladder with needle and syringe, then applying the clamps and completing the operation as a lateral anastomosis.

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CHAPTER 71

VERMIFORM APPENDIX

By A. V. Partipilo

Anatomy.—The term vermiform appendix is derived from the Latin word vermiformis, from vermis—worm, plus forma—shape. Appendix is derived from the Latin word appendere, meaning to hang upon, an appendage. Therefore, vermiform appendix may be defined as a worm-shaped appendage of the cecum. It hangs down from the medial and posterior part of the cecum about 1 to 1½ inches from the ileocecal orifice. Its situation from that point is variable. If has been found in almost all possible positions and situations in the abdomen. Normally it is suspended from the cecum in three directions:

1. Over the brim of the pelvis.
2. Upwards and medially toward the spleen.
3. Upwards behind the cecum.

The size of the appendix is extremely variable. No two appendices are of the same length and breadth. The average length has been found to be about 3 inches. Cases have been reported of appendices being nine inches long and as short as ¾ inch. The average breadth of the appendix is about ¼ inch. Its lumen communicates with the cavity of the cecum at its medial posterior aspect. At this point the mucous membrane may serve the purpose of a valve (Gerlach's valve). The communication between the two organs varies considerably. For want of function the appendix undergoes degenerative changes so that at about middle life it has the appearance of a hard fibrous cord. Its lumen is hardly noticeable. This type is clinically considered as obliterative appendicitis.

During embryological development the appendix receives the same structures that go into the development of the cecum. This includes the longitudinal and circular muscles, the nerves, and glandular elements. In the cecum the longitudinal muscle separates into three bands. In the appendix they are intact and not separated. From the base of the appendix the longitudinal muscle passes over the cecum into three layers, called the tæniæ coli, which constitute one of the distinguishing features between the large and the small bowel. They are of practical value in locating the appendix.

In the great majority of cases the appendix has a well-developed mesentery. It extends from the base to the tip of the appendix, and becomes attached to the inferior part of the mesentery of the terminal ileum. The mesentery to the appendix, called the meso-appendix, is the medium through which the appendiceal vessels reach the organ. In some cases the meso-appendix may be so small as to be almost negligible; in other cases it has been found to be quite extensive. When the meso-appendix is long, the appendix may be found on the left side of the abdomen or high up close to the gall bladder. When the mesentery is short, the appendix usually lies on the anterior, posterior, lateral, or medial side of the cecum. The position of the appendix may be the determining cause for it to become involved secondarily in

an inflammatory process in the pelvis, gall bladder, stomach, or any organ in the abdomen.

The blood supply to the appendix comes from the ileocolic artery which is a branch of the right colic artery. From its point of origin the appendiceal artery passes behind the terminal ileum where it enters the meso-appendix. During its course in the meso-appendix the artery sends several branches across to the appendix which finally end in its tip.

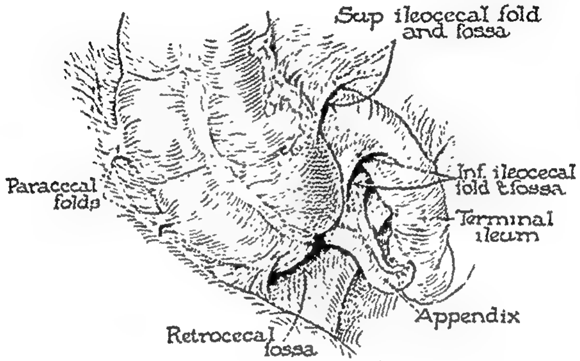

FIG. 609.—Fossæ and folds of the cecum.

METHODS OF LOCATING THE APPENDIX

By Following the Taenia-Coli.—The appendix can be located during an operation by following the anterior taenia coli of the colon and cecum down to its termination at the base of the appendix. Locating the appendix by this method implies a knowledge of the structure of the large intestine, and the ability to differentiate the large from the small bowel. The following are the most important differences:

1. The large bowel has taenia coli, three in number, which converge at the base of the appendix. They are found wanting in the small intestine, because the longitudinal muscle spreads evenly over the entire circumference of the bowel.

2.

tions

coats, thereby causing the bowel to pucker (sacculate). This condition can be demonstrated easily by dissecting the bands, which results in straightening of the bowel. It must be emphasized that when the large bowel is distended with gas the sacculations may not be so evident, or may be entirely absent.

3. Appendices epiploic on the large intestine are projections of peritoneum containing variable amounts of fat. They are found in the entire length of the colon with the exception of the rectum. In the cecum and the ascending colon they are somewhat broad and flattened, whereas, in the sigmoid they are elongated and globular. This is one of the distinguishing features between the ascending colon

and the sigmoid. It is also of practical importance, because at times the sigmoid may migrate in the right fossa, and may be mistaken for the ascending colon. The finding of long globular appendices epiploic indicates that the sigmoid instead of the ascending colon has been found. By looking for and examining the character of the appendices epiploic in a routine manner, much time can be saved in locating the appendix.

4. The ascending colon which is found in the right lateral body wall has a mesentery that is short and often absent, while the mesentery of the small intestine is well defined and extends from the second left lumbar vertebra to the right iliac fossa. When the surgeon is in doubt, he may locate the bowel by tracing the mesentery to its base; the mesentery of the small intestine is directed toward the spine, while that of the large bowel is toward the lateral body wall.

5. Other differences are: the lumen of the large intestine is larger than that of the small intestine; the large bowel has a bluish-color; and the large bowel is relatively thicker than the small bowel. These differences are not of much practical value because they are not constant.

Direct Method.—A direct and simple method of locating the appendix consists in passing the left hand within the abdomen until the index finger touches the anterior superior spine. The fingers are then spread apart so that the middle finger hugs the brim of the pelvis. After that the fingers are carried downward and posteriorly across iliac fossa where the index finger hooks the cecum while the middle finger hooks the terminal ileum. The cecum and ileum are then carried inward and forward (toward the abdominal incision) thus exposing the posterior surface of the cecum and the base of the appendix which lies between the index and middle fingers.

Incision.—Choosing the right incision for appendectomy entails a consideration of many factors. To make one routine incision to fit every case is unjustifiable, for every case presents a different problem. The selection of the type of incision should be based upon three factor, namely, the age, the sex of the patient, and the inflammatory stage of the diseased appendix. For boys below the age of 18 with symptoms of acute appendicitis, McBurney's incision is ideal. This is due to the fact that in this age group, acute appendicitis is the most predominant cause of an acute abdomen. If the appendix is ruptured, the original incision may be closed and drainage established through a stab wound made over the crest of the ilium. After the age of 18, errors in diagnosis are more frequent, hence the type of incision should be one which will permit exploration of the abdomen. For this reason, a McBurney incision is not adequate. A vertical incision permits exploration of the upper and lower abdomen if the appendix is not the cause of the acute abdominal symptoms. In chronic appendicitis the incision should always be vertical in order to explore the abdomen adequately.

For girls below the age of fourteen years who show symptoms of acute appendicitis, McBurney's incision is preferred. Past this age, the surgeon, when choosing the incision, must take into consideration the possibility that an acute salpingitis or a twisted ovarian cyst may be the cause of the acute abdominal symptoms. For chronic appendicitis, a McBurney's incision should not be made since it does not allow for sufficient exploration of the pelvis. Hence, a paramedian or vertical midrectus incision is preferable (see Chapter on "Abdominal Incision," p. 432.)

Extirpation of the Appendix.—After the appendix has been found and freed from its adhesions, the meso-appendix is grasped at the tip of the appendix with a forceps. The appendix is raised and brought out to the abdominal incision. If the

meso-appendix is short, it is ligated en-masse by passing a No. 1 plain catgut ligature through the meso-appendix at the base of the appendix; if long, it is ligated with two or more interlocking knots. In either case the ligature is tied and the ends are left long and secured in a ligature forceps. The meso-appendix is now cut between the ligature and the appendix. Instead of using ligatures the meso-appendix may be grasped with forceps and incised. If the mesentery is long, troublesome

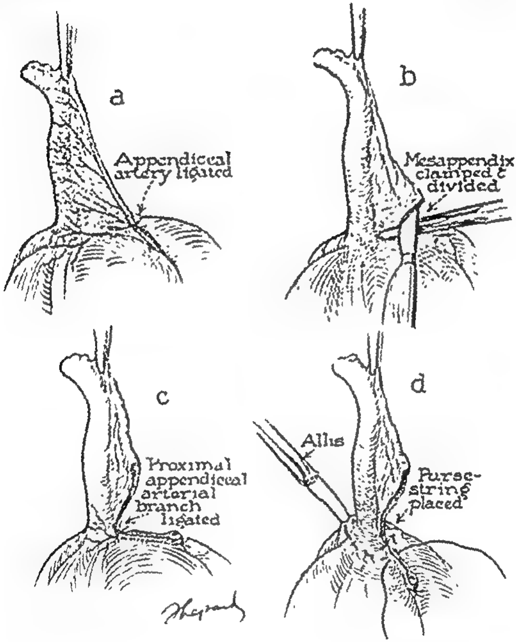

Fig. 810. [illegible]

bleeding can be averted by taking small bites with forceps until the entire mesentery has been grasped and incised. Each crushed portion is then ligated. Before proceeding, make certain that all bleeding points are ligated, especially at the base of the appendix where a posterior branch may be present. To forestall postoperative bleeding from the posterior branch, the author applies a submucous stitch on the cecum close to the base of the appendix.

Two clamps are now applied: one grasping the appendix at the junction with the cecum and the other about ½ inch distal. The proximal forceps is removed and a ligature is tied around the crushed grooved surface formed by the clamp (Fig. 611). A purse-string suture of silk is now inserted around the base of the appendix. The appendix is amputated distal to the ligature with either cautery or scalpel. The amputated appendix and scalpel are discarded and the stump of the appendix is cauterized with phenol and alcohol. To invert the stump a tissue forceps is placed underneath the purse-string, grasping and depressing the stump while the purse-string is being tied (Fig. 612 *b*, *c*). By placing the tissue forceps beneath the purse-string, it should be noted that after the suture is tied the forceps is outside of the purse-string. After the purse-string is tied, a few interrupted sutures may be applied to reinforce the inner suture. If the meso-appendix is long, it can be stick-tied to the invaginated portion of the cecum.

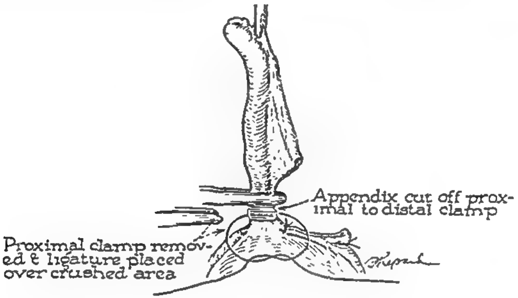

FIG. 611.—Method of ligating the stump of the appendix in the groove formed by the crushing forceps.

There are many methods of inverting the stump, the aforementioned being the most popular. However, it must be emphasized that the stump is not actually inverted by this method, because it is impossible to obtain a complete inversion with the ligated stump. In reality, the stump is buried within the walls of the cecum. To obtain an actual inversion the technique is as follows: after the appendix has been detached from its mesentery, a purse-string suture is applied and the appendix is then extirpated in between two clamps. The amputated appendix is discarded, and the stump held in the clamp is carbolized with phenol and alcohol. A tissue forceps is placed beneath the purse-string suture and grasps the stump of the appendix being held with the clamp. The latter is discarded and the stump is depressed with the tissue forceps while the purse-string is being tied (Fig. 612). The operation is then completed similarly to that described above.

If the appendix is bound down with adhesions, or if there is difficulty in reaching the tip of the appendix, it may be impossible to remove the appendix from the tip downward. Under such circumstances the appendix may be removed in a retrograde manner. This is accomplished by severing the appendix between the two

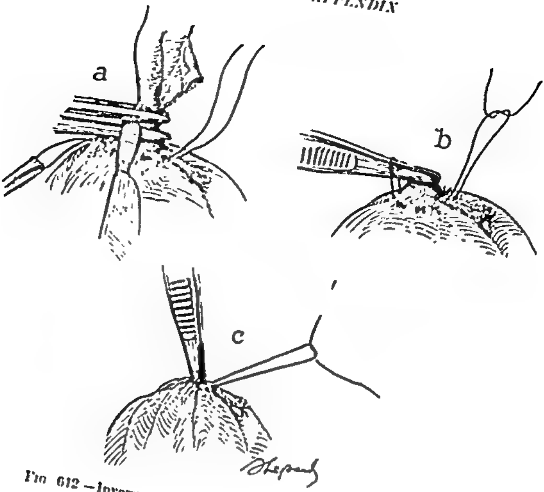

FIG 612 —Inversion of the appendiceal stump without ligation.

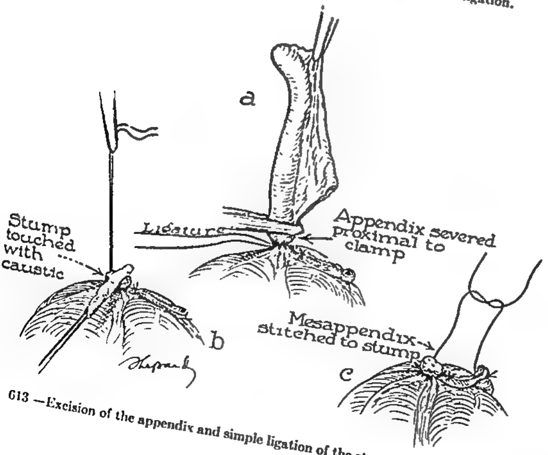

FIG 613 —Excision of the appendix and simple ligation of the stump without inversion

clamps; the stump is inverted with a purse-string suture in the usual manner, and meso-appendix is then divided from the base to the tip of the appendix.

There are surgeons who do not invert or bury the stump, but simply tie a ligature around the base after the appendix is freed from the mesentery (Fig. 613). This technique should not be done as a routine. However, in the presence of an edematous, infiltrated, or gangrenous appendix, a purse-string suture is oftentimes impracticable; a simple ligature without invagination of the stump will suffice. The meso-appendix should be sutured to the stump. This technique is also applicable when an abscess is present. In such cases the appendix is extremely friable, and the application of any kind of suture will easily tear out, hence, a simple ligature tied at the base is the most that can be done.

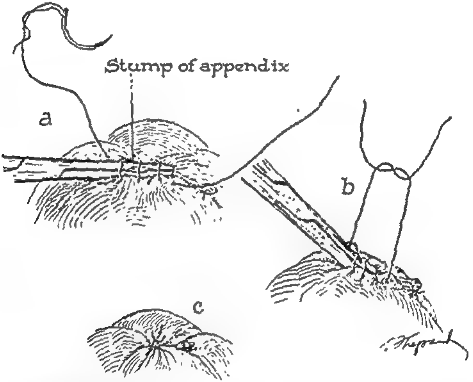

Fig. 614.—Author's method of inverting the appendiceal stump, (a) Kerr-Parker stitch applied over a Carmalt forceps which holds the unligated stump, (b) the forceps is removed and the suture is pulled taut; (c) the suture is tied.

Figure 614 illustrates the author's method of inverting the stump of the appendix. It is essentially similar to the Kerr-Parker method of inverting the end of the bowel in a lateral anastomosis. This method is extremely helpful when the appendix is retrocecal or deeply imbedded.

The question of drainage after an appendectomy is a controversial subject. In the face of free pus, adequate drainage must be instituted. If actual pus is not present, but free fluid appears within the abdomen, drainage is not so important. For a further discussion of this problem refer to the chapter on the peritoneum.

QUESTIONNAIRE

1. Define vermiform appendix.
2. What are the normal positions of the appendix?
3. What is Gerlach's valve?
4. What are taeniae coli?
5. Give the origin [illegible]
6. [illegible]
7. [illegible]
8. [illegible]
9. [illegible]

CHAPTER 72

SURGERY OF THE LARGE BOWEL

By Clement L. Martin

GENERAL CONSIDERATIONS

Progress in colonic surgery has been remarkable in the twenty-seven years since the first edition of this book was published. Antibiotic drugs are the most important cause of this in the last ten years but increased experience based on a knowledge of electrolytes, the application of biochemical information, *e.g.*, potassium depletion and other specific chemical requirements, have been of great consequence. Generally there is a better understanding of obstruction and its physiologic effects, as is true of anemia, protein and vitamin requirements, particularly vitamin K, and a knowledge of the patient as an organism and not merely the site of an operation.

The bowel wall below an obstructing carcinoma is often edematous and chronically inflamed, that above distended and thinned. In the partially obstructed bowel the wall both above and below may be inflamed and thickened, with telangectases, prominent vessels and edema, and not infrequently with inflammatory thickening and palpable or visible glands in the adjacent mesentery.

The chronically thickened and infected bowel found in chronic ulcerative colitis or a carcinoma may, on moderate handling, exude serum containing virulent bacteria. Vigorous manipulation of the cancer-bearing segment of bowel may produce metastases. Some surgeons now tie the principal vessels before handling a neoplasm of the colon.

Relief of the subacute obstruction and its attendant local effects is of primary importance. Preoperative preparation should provide this relief, as well as that of dehydration and anemia, if present, and protein deficiency if the serum protein is less than 6 gm. per 100 cc. An adequate carbohydrate ingestion to provide an hepatic glycogen reserve, and supplying vitamin K if the prothrombin time is low, are essential. The necessity of adequate preparation is comparable to that in genito-urinary disease: carcinomatous bowel must be cleaned out, and kept reasonably empty to permit the hypertrophied and dilated wall to recover and the infection in and about the lesion to diminish. The patient's physical reserve should be evaluated on the basis of the general physical examination and laboratory findings. Colonic surgery is more difficult in the obese, the reparative process in fat is slow and infection more likely. Pelvic resections are easier in women because of the broader pelvis. Anterior resection of the rectum is less difficult in thin than in fat men.

The colon is less vascular than the small intestine and rapid repair cannot always be anticipated as in surgery of the small bowel. A less rich blood supply, edema and infection, actually or possibly present in the wall, makes it desirable to avoid undue peristalsis and distention from gas during the first few postoperative days. Failure to heal at the suture line is more often due to leakage from infection or faulty suturing rather than lack of vascularity.

Suturing must be exact and interrupted sutures are preferred. They must be placed close together in a contracted bowel lest leakage occur when it expands, and not too tight so that they cut through, or so loose they fail to hold the opposed surfaces securely. Silk, linen, cotton or wire sutures are matters of individual preference. The Kerr-Parker "aseptic" anastomosis and the use of the Rankin or Partipilo clamps are described later. Clamp resection has been largely discontinued because of effective preoperative medication.

In open anastomoses, whether a single row of interrupted (generally mattress) sutures or an inner row of continuous suture and an outer of interrupted, or a double row of interrupted sutures is used, is also a matter of personal opinion. Most surgeons feel it is safer to use two rows, at least one of which, usually the outer, is of metal, silk or other fiber. Again, whether a mattress (Halstead) or Lembert is used depends upon local factors and especially upon personal preference. Wangensteen and others have shown that a single row of mattress sutures are adequate "from the stomach to the rectum." This is not recommended to the surgeon whose resections are few.

The choice of operation in surgery of the colon is dependent upon the following factors: the portion of the bowel involved, whether it has a full mesentery and is ordinarily movable; the general character and detail of blood supply to the part; the presence or absence of obstruction, either partial or complete; and the greater consistency of the content of the left half of the colon.

The number of procedures offered for a single lesion may be confusing. For instance, carcinoma above the hepatic flexure, after liberal mobilization, may be treated by Lahey-Mikulicz, Rankin obstructive resection or ileo-colostomy. Appreciating the fundamental principles concerned, decision is based on the physical status of the patient and the procedure that the surgeon can do most satisfactorily and safely. But obstructive resections and the Lahey-Mikulicz operation, like clamp operations are not so much used, because of better control by antibiotic medication of the intestinal flora. Open anastomosis is now relatively safe.

Cole[1] lists the following as axioms of colonic surgery: Imbalances such as anemia, hypoproteinemia, malnutrition, etc. must be corrected before resection is performed: the chief cause of peritonitis is leakage, after, not soilage at operation; decompression of the small intestine does not relieve obstruction of the colon; complete obstruction usually requires immediate operation, *i.e.*, colostomy, with resection later. Obstructive resection is now permissible in partial obstruction; a double barrelled loop colostomy is preferable to a tube—single barrel—colostomy; since introduction of antibiotic medication, open anastomosis may be considered safe; aged patients do not tolerate resection as well as younger patients and therefore need better preoperative care, antibiotics are not substitutes for sound surgery; no one operative procedure is applicable to all cases; mortality improves with perfection of a method.

The procedures here described are those generally accepted, most used, or which we think are proven the best in their field. Numerous others are illustrated and defined in current texts, and in the hands of their authors or protagonists have merit. However, some colonic operations depicted in current texts are already obsolete; a choice is impelled and selections are made. Consult current surgical texts and the recent literature as to pathology, diagnosis, operability, end-results

[1] Cole, W. H. Cancer of the Colon, Ill. Med. J., May, 1947

and related subjects, as well as for more complete descriptions of the many operations available. Colonic operations should not be undertaken with limited knowledge or training or inadequate facilities.

Of necessity, because of limitations of space in any work on surgical technique, a didactic style is often used; this in spite of the fact that the subject does not lend itself to dogmatic statement.

ANATOMICAL CONSIDERATIONS

Embryologically, the large bowel has its origin on the left side of the abdomen, the cecum migrating from the left to the right. During the third month the cecum is found beneath the stomach and at a later date over the liver; subsequently it moves over the right kidney, and finally just before birth, it descends into its normal position in the right iliac fossa. Except in rare instances, the descent and rotation

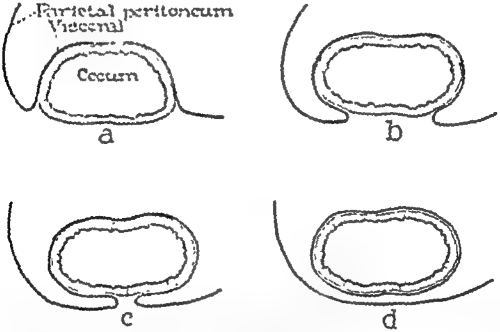

FIG. 615.—Cross-sections in the region of the cecum to show the variations of the peritoneal coverings on the cecum; (a) the posterior surface is widely exposed, and the cecum is bound down; (b) the posterior is less exposed, with a suggestion of a mesentery; (c) the cecum is entirely covered with peritoneum and is suspended by a mesentery, (d) the cecum is entirely free and unattached.

may not occur; however, in such cases the cecum will be found in the left iliac region. As a result of congenital malformations or arrest, the cecum may be found in all possible locations between the left and right iliac fossæ.

The large bowel is from 4½ to 5 feet long, and is arched in the form of an inverted U or M. It is composed of the following subdivisions: cecum; ascending colon; hepatic flexure; transverse colon; splenic flexure; descending colon; pelvic colon. It terminates in the region of the third sacral vertebra, in the rectum.

Cecum.—The cecum is found entirely within the right iliac fossa. However because of abnormal development, it may lie high up in the right lumbar region, or may overlie the brim of the pelvis. It has been encountered in all situations within the space between the right kidney and the left ovary. The mobility of the cecum is dependent upon its peritoneal covering. In the great majority of cases it is

completely invested with a visceral layer, thus becoming free and unattached. (Fig. 615). *In this instance, it is extremely mobile; hence the term "mobile cecum."* In a few individuals the posterior surface is exposed to a varying degree. The extent of this exposure determines the mobility of the cecum. When the cecum is widely exposed, it is firmly bound down, and the appendix may be found retrocecally.

The cecum rests on the iliopsoas muscle; hence, in appendicitis, especially in the retrocecal appendicular abscess, the thigh becomes flexed as a result of irritation of the psoas muscle. *This is nature's attempt to relieve the muscle tension, thus decreasing the pressure of the lumbar nerves within the psoas fascia.* Alternately flexing and extending the right limb elicits pain. This is the "psoas test" for acute appendicitis.

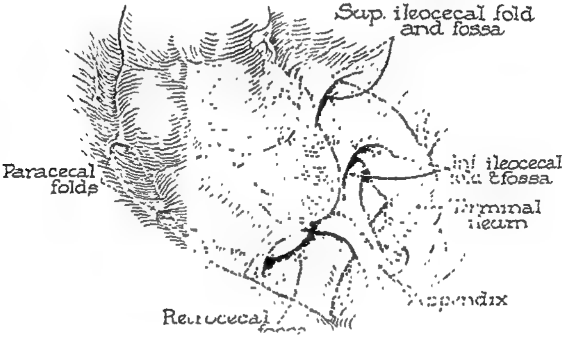

FIG. 616.—Fossae and folds of the terminal ileum and cecum.

On the posterior-medial wall of the interior of the cecum is found the ileocecal *orifice, guarded by the ileocecal valve.* The latter is formed by the invagination of the wall of the ileum into the cecum producing two folds which lie above and below the orifice. Only the circular muscular fibers of the ileum take part in the formation of the valve; whereas, the longitudinal muscle and peritoneum are stretched on the exterior of the cecum as a result of the inversion, thus serving to preserve the fold and the formation of the valve (Cunningham). This anatomical arrangement is a factor in the production of intussusception.

There are three cecal fossæ near the termination of the ileum and the cecum (Fig. 616). The superior ileocecal fossa extends between the ascending colon and the terminal ileum. The inferior ileocecal fossa lies between the ileum and the cecum. It is formed by the ileocecal fold which extends from the terminal ileum to the cecum and the mesentery of the appendix. This fold is called the bloodless fold of Treves. The retrocecal fossa is found on the posterior surface of the cecum. It is the largest and the most constant of the three fossæ.

The blood supply to the ileocecal appendicular segment is derived from the ileocecal artery. This artery arises by a common trunk with the right colic, or may

arise as a distinct branch of the superior mesenteric artery. It originates at the right side of the superior mesenteric artery below the third portion of the duodenum, then it runs downward and to the right, across the ureter and psoas muscle, behind the peritoneum, toward the ileocolic angle. The ileocolic artery divides into five branches: (1) appendicular; (2) anterior cecal; (3) posterior cecal; (4) colic; and (5) ileal branch. The anterior and posterior cecal arteries supply corresponding parts of the cecum. The ileal branch anastomoses with the terminal branch of the superior mesenteric artery. The appendicular branch passes behind the terminal ileum, the meso-appendix and runs along the free border of this mesentery to reach the tip of the appendix, where it enters. The colic branch runs upward, along the ascending colon, to anastomose with the right colic artery.

Ascending Colon.—The ascending colon is continued from the cecum in an upward and posterior direction, until it reaches the inferior border of the liver, where it bends forward and to the left, giving rise to the hepatic flexure of the colon. The posterior surface of the ascending colon lies upon the iliacus and quadratus lumborum muscles and the lower pole of the kidney. It is connected to the muscles by a loose areolar tissue, and is separated from the kidney by the perinephric fascia.

The blood supply for the ascending colon arises from the right colic artery, a branch of the superior mesenteric artery, but it may come chiefly from the ileocolic artery.

Hepatic Flexure.- The hepatic flexure is situated between the terminal part of the ascending colon and the transverse colon. Its direction is forward and to the left, until the descending portion of the duodenum is reached, where it is continuous with the transverse colon. It lies on the anterior surface of the right kidney and over the third part of the duodenum. Its medial surface is in contact with the gall bladder. The blood supply is derived from the anastomotic branches of the middle colic and the right colic arteries.

Transverse Colon.—The transverse colon runs transversely from the hepatic to the splenic flexures. As it crosses the abdominal cavity it takes a downward curve, its lowest part usually reaching the umbilicus. In many cases it may reach a much lower level. The first part of the transverse colon is attached to the descending part of the duodenum and to the head of the pancreas by a short mesentery. At the left extremity it is attached to the tail of the pancreas. Between its two extremities the transverse colon is suspended from the posterior wall by the mesocolon. Anteriorly, it is covered by the greater omentum (gastrocolic ligament) through which it is loosely connected to the stomach. The blood supply for the transverse colon is derived from the middle colic and left colic arteries.

Splenic Flexure.—The splenic flexure is situated deeply underneath the ribs and is overlapped partially by the stomach. It is a continuation of the transverse colon. The terminal portion of the transverse colon is directed cephalad, posteriorly, and to the left until the base of the spleen is reached, when it bends sharply, forming the splenic flexure, and the latter then takes a downward direction to become continuous with the descending colon. The splenic flexure is held in position by its attachments to the pancreas, spleen, and to the diaphragm by the phrenocolic ligament. Sectioning this ligament permits mobilization. Posteriorly, it is in relation to the anterior surface of the left kidney.

Descending Colon.—The descending colon begins at the splenic flexure, passes downward along the left side of the abdomen, and ends in the lumbar region by passing into the sigmoid colon. The first portion is in contact with the lateral

border of the left kidney. The descending colon is covered with peritoneum on its sides and anterior surfaces; the posterior, being devoid of peritoneum, is connected to the body wall by loose areolar tissue. In some cases the peritoneal covering is complete and consequently the bowel is supplied with a mesentery. Its blood supply is derived from the left colic artery, a branch of the inferior mesenteric artery.

Sigmoid Colon.—The sigmoid colon may be divided into two parts, (1) iliac, and (2) pelvic colon. The iliac portion of the sigmoid has a mesentery sometimes rather limited and lies upon the iliopsoas muscle. Beginning at the level of the crest of the ilium it passes downward and medially in front of the iliacus muscle, and ends at the medial border of the psoas major muscle where it dips into the pelvis and becomes continuous with the pelvic colon. The latter is suspended from the posterior wall by a well-developed mesentery which permits of considerable movement. The pelvic portion of the sigmoid colon begins at the medial border of the psoas major muscle and passes upward and medially along the mesial side of the external iliac vessels, and, after crossing the hypogastric vessels, it turns downward to end at the level of the third sacral vertebra, where it becomes continuous with the rectum. The sigmoid colon derives its blood supply from sigmoidal branches of the inferior mesenteric artery. In its passage into the pelvis, the sigmoid colon crosses the left ureter, the internal testicular or ovarian vessels, the femoral nerve. the external iliac vessels and the hypogastric (internal iliac) vessels.

BLOOD SUPPLY

There is much variation in this vascular anatomy. Summarizing the conclusions from the studies of Rankin[1] and Steward:

A. In regard to *ileocolic* artery, it may be concluded: (1) the existence of the artery is constant; (2) its course is toward the ileocolic valve; (3) the terminal branches (colic, appendicular, etc.) vary in their origin.

B. The *right colic* artery is the most inconstant of the colic arteries. It originated from the superior mesenteric in 40 per cent (of 40 cases) from the middle colic in 30 per cent, from the ileocolic in 12 per cent, whereas in 18 per cent there was no artery that corresponded in course or distribution to the right colic artery. It may be concluded that the right colic artery is extremely variable in presence, origin and size.

C. The *middle colic* artery, the first branch of the superior mesenteric, runs to the right between the layers of the transverse mesocolon, then divides, anastomosing with the right and left colic branches; it supplies chiefly the transverse colon. It does not occur constantly; it varies in number of branches. Through large branches or by accessory middle colic arteries it supplies the left half of the colon and splenic flexure in 37 per cent of cases.

D. The *left colic* artery, the first branch of the inferior mesenteric, passes transversely to the left and divides into an ascending and descending branch. There is marked variation in the size, course and distribution of its branches; in most cases the ascending branch extends above the splenic flexure to the transverse colon. This artery is almost invariably present.

The *marginal* artery is generally present and surgeons successfully ligate individual arteries as the occasion demands and rely on vascularization through the

[1]RANKIN, FRED W., & GRAHAM A. STEPHENS, Cancer of the Colon and Rectum. CHARLES C THOMAS, Springfield, Ill. 1939, p. 12 et seq.

marginal artery. However, it was absent in 5 per cent, and in these the failure of anastomosis was between the right colic and ileocolic arteries.

Between the middle colic and the left colic arteries there was no failure of anastomosis in more than 100 specimens in which this particular area was examined.

LYMPH GLANDS OF THE COLON

The lymph glands of the colon are divided by Jamieson and Dobson into four regional groups: (1) Ileocolic; (2) Middle colic; (3) Left colic; (4) Inferior mesenteric. The lymph nodes in each region are in four separate but intercommunicating groups: (A) the epicolic glands situated on the wall of the bowel and in the appendices epiploicæ; (B) the paracolic lymph glands lying posteriorly and medially to

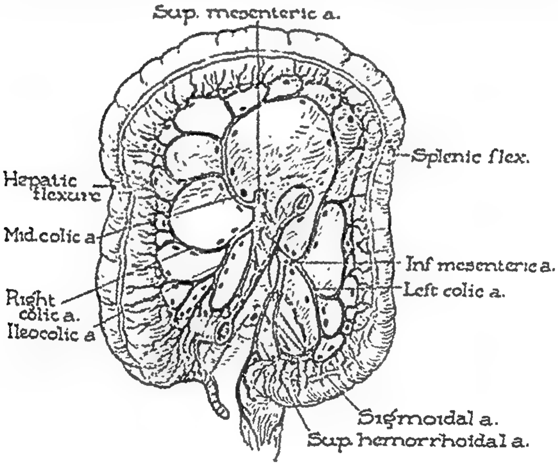

FIG. 617.—Lymph nodes and blood vessels of the large bowel.

the ascending, descending and iliac portions of the colon between the intestine and the arterial arcades and on the arcades, along the upper border of the transverse colon and on the mesenteric side of the pelvic portion of the sigmoid; (C) the intermediate group of lymph glands found along the ileocolic, right colic, middle colic, inferior colic arteries and their branches and about the trunk of the arteries midway between their origin and the bowel; (D) the central lymph glands (main nodes) lying along the superior and inferior mesenteric arteries, near their origin (Fig. 617).

LYMPH GLANDS OF THE RECTUM

Anatomists divide the lymph glands of the rectum into three groups: (1) intramural (2) intermediary and (3) extramural.

1. *Intramural Lymphatic System.* These lymph glands are found in the wall of the bowel and consist of two networks, one situated in the submucous, the other in the muscular layers of the bowel. The two intercommunicate by means of short channels which pass through the circular muscle. The submucous network is continuous with that of the pelvic colon and below it with a network in the anal canal. The latter communicates with the lymph glands of the perianal skin. The intermuscular network also communicates with the pelvic colon, and below with the lymphatics of the external sphincter muscle.

2. Intermediary Lymphatic System. This system consists of a network of glands which lie under the peritoneal layer of the bowel. When the peritoneum is deficient, a lymph sinus is found in the perirectal fat. The collecting stems from the intermediary network empty into this lymph sinus.

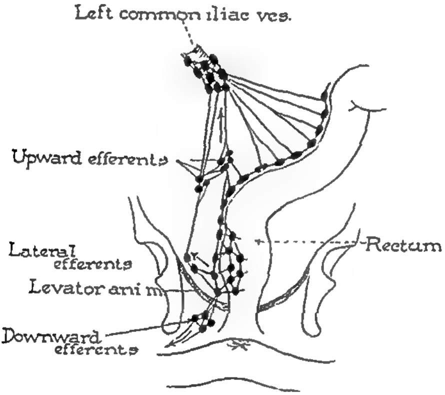

FIG. 618.—Extramural lymphatics of the rectum. Arrow points to the direction of the efferents (modified after Miles).

3. Extramural Lymph System. These glands are the most important of the lymphatic systems. According to Miles, "The collecting stems from the lymph sinus form an extensive plexus and enter into relation with the anorectal glands of Gerota, which are scattered over the surface of the rectum among the branches of the superior hemorrhoidal vessels. The efferents from this plexus and from the anorectal glands pass to their ultimate destination in three directions, namely, downward, laterally and upward." The downward efferents pass across the ischiorectal fossa to the internal iliac vessels. The lateral efferents enter the plexus between the levator ani muscles and the pelvic fascia from which they pass to the obturator gland, thence to the internal iliac glands. The upward efferents accompany the superior hemorrhoidal vessels to the pelvic mesocolon, and finally to the glands situated at the bifurcation of the left common iliac artery. Some of the

efferents enter in relation with the paracolic glands at the region of the superior mesenteric vessels.

CONGENITAL MALFORMATIONS

Among the congenital deformities the displacements of the colon (dystopia coli), and the extreme dilatation of the colon (Hirschsprung's disease), are of primary importance to the surgeon, as well as to the medical confrere. Dystopias coli, producing alarming symptoms, are not very frequent. Most of them have been observed at autopsy, and more recently during roentgenographic examinations for suspected lesions elsewhere in the gastro-intestinal tract.

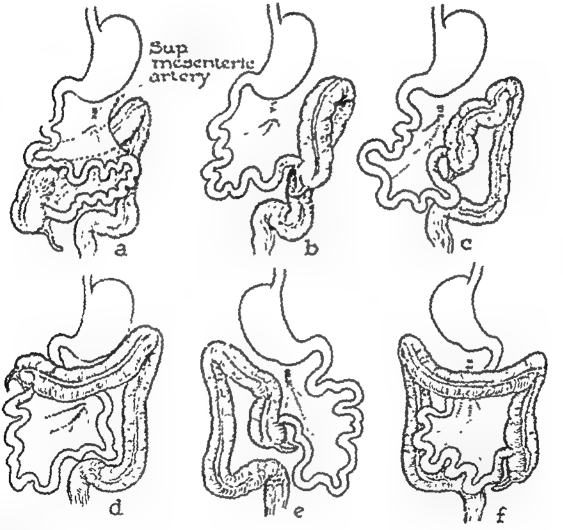

FIG. 619.—[illegible]
torsion—[illegible]
with norr[illegible]

In most individuals, malformations of the colon do not cause any noticeable dysfunction, but in a small percentage of cases they may cause obstruction by a volvulus. Hecker[1] believed that most of the colonic ectopias, excepting those due

[1] The Malformations and Displacements of the Large Intestine and their Surgical Importance, Am. J. Surg. 1926.

to excessive length, result either from insufficiency of normal rotation of the primitive intestinal loop, or from inverse rotation or from deficient fixation. The following is a classification of colonic dystopias based upon the conclusions of Hecker:

1. Faults of Rotation.—A. Absense of all rotation. The large bowel is in a plane posterior to the small intestine. The small intestine and its mesentery lie anterior to the large bowel. The duodenal-jejunal flexure is situated above the origin of the superior mesenteric artery. Absence of any rotation predisposes, by the retromesenteric position, to a dilatation of the colon, and to an acute obstruction.

B. Insufficiency of normal rotation (anti-clockwise):

1. Sinistro-colia of first degree with normal rotation. After rotation of 90° the small intestine is found to the right and the large intestine to the left; the duodenal-jejunal flexure is to the right and on the same level as the superior mesenteric artery.

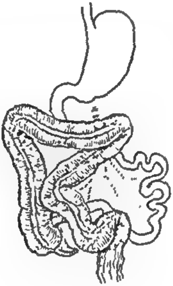

FIG. 620.—False dextro-colia with normal rotation due to ptosis of the splenic flexure

2. Sinistro-colia of second degree with normal rotation of 90 to 180°. In this condition after a rotation of 180° the colon is still on the left, but the ileocecal coil touches the umbilical region. The splenic flexure, which does not make more than 135° of rotation, remains in the left hypochondrium. The duodenal-jejunal angle lies below the mesenteric root.
3. Dystopia of third degree with normal rotation of 180 to 270°. After 270° rotation ceases. The cecum is found sub-hepatic, the small intestine on a posterior plane in relation to the transverse colon, the duodenal angle is definitely in its normal place, *i.e.* to the left of, and on the same level, as the origin of the superior mesenteric artery.
4. A cecal ectopia may persist in adults due to insufficient descent of the cecum.

The sinistro- and destro-colias, presenting acute angulations, predispose to an acute occlusion, to a distension with stercoral stasis above the obstructing angle, and especially to a volvulus of a movable loop.

C. Insufficiency of inverted rotation (clockwise). In cases of inverse rotation, there are, after the viscera have regained the abdominal cavity, dispositions described for normal rotation. They are the following:

1. Dextro-colia of first degree with inverted rotation between 0 and 90°.

2. Dextro-colia of second degree with inverse rotation between 90 and 180°.
3. Dystopia of third degree with inverted rotation between 180 and 270°.
4. Insufficiency of descent of the cecum. The termination of the descent of the cecum in the left fossa iliaca produces a situs inversus partialis inferior.

INSUFFICIENCY OF FIXATION

A. Ptosis of fixation of the colonic flexures.

1. Ptosis of the splenic flexure: (*a*) false dextro-colia with normal rotation; (*b*) false sinistro-colia with inverted rotation. Absence of fixation of the splenic flexure predisposes to a volvulus, however, this condition is rare.
2. Ptosis of the hepatic flexure: (*a*) false dextro-colia with inverted rotation; (*b*) false sinistro-colias, due to ptosis of the splenic flexure, are easily recognized, while those due to ptosis of the hepatic flexures with dextro- and sinistro-colias, are difficult to diagnose.

As previously stated faults and arrest of rotation, as well as ptosis, do not ordinarily evidence their presence by dysfunction and thus do not produce symptoms. Surgery may be necessary for a complication such as volvulus. Sigmoidopexy is sometimes done in prolapse of the rectum, but it is of uncertain value, even when the real cause, which is a hernia of the bowel through the pelvic fascia and levator muscle, is treated.

to excessive length, result either from insufficiency of normal rotation of the primitive intestinal loop, or from inverse rotation or from deficient fixation. The following is a classification of colonic dystopias based upon the conclusions of Hecker:

1. **Faults of Rotation.**—A. Absense of all rotation. The large bowel is in a plane posterior to the small intestine. The small intestine and its mesentery lie anterior to the large bowel. The duodenal-jejunal flexure is situated above the origin of the superior mesenteric artery. Absence of any rotation predisposes, by the retromesenteric position, to a dilatation of the colon, and to an acute obstruction.

B. Insufficiency of normal rotation (anti-clockwise):

1. *Sinistro-colia of first degree with normal rotation. After rotation of 90° the small intestine is found to the right and the large intestine to the left; the duodenal-jejunal flexure is to the right and on the same level as the superior mesenteric artery.*

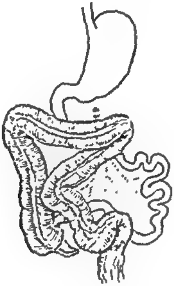

FIG. 620.—False dextro-colia with normal rotation due to ptosis of the splenic flexure.

2. Sinistro-colia of second degree with normal rotation of 90 to 180°. In this condition after a rotation of 180° the colon is still on the left, but the ileocecal coil touches the umbilical region. The splenic flexure, which does not make more than 135° of rotation, remains in the left hypochondrium. The duodenal-jejunal angle lies below the mesenteric root.
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CHAPTER 73

DISEASES OF THE LARGE BOWEL

By Clement L. Martin

GRANULOMATOUS DISEASES

Hyperplastic tuberculosis is rare; it is nearly always in the cecum; several cases have been reported in the sigmoid and rectum. It is important chiefly because it is the one kind of intestinal tuberculosis in which surgery offers a cure. It is apparently a primary infection from direct invasion of the mucosa by *mycobacteria tuberculosis* ordinarily: *Brown and Sampson found 76 of 100 cases to be primary* infections[1], thus the lungs in three-fourths of their cases were not involved. "The lesion is essentially proliferative, while ulceration, which often or even usually is present, is not as a rule a conspicuous feature . . . the walls of the affected gut become very thickened and hardened, all the coats being involved. In the mucosa and submucosa there is great overgrowth of lymphoid tissue with much fibrosis, edema and lymphocytic infiltration. The muscular coat is hypertrophied and invaded by fibroblasts and lymphocytes, while the subserosa is also thickened and fibrosed and in both of these sites nodules of lymphoid tissue may make their appearance . . . The intestinal enlargement may be continued into a dense fibro-adipose mass in the mesocolon or mesentery." [1]

The incidence is higher in men than in women, and 80 per cent of the largest group reported were less than forty years of age, 51 per cent were under thirty.

A mass is often palpable in the right lower abdomen. Obstruction from progressing fibrosis, edema and resultant stenosis occurs, usually at the ileocecal juncture but may develop in the ascending colon.

Diagnosis is dependent upon roentgenologic examination, a barium enema disclosing the lesion, which can usually be differentiated from cancer.

A resection of the right colon and ileostomy is the ordinary procedure here; some surgeons resect the ascending colon and anastomose the ileum to the hepatic flexure, when the area of colonic involvement permits. A right hemicolectomy is ordinarily more certain of eliminating involved glands and mesentery, and lessens *the hazard of a doubtful blood supply to the remaining bowel.*

Regional colitis is similar to the regional (segmental) ileitis occurring in the terminal ileum. It involves any part of the colon except the rectum, more often the right side, and progresses in either direction, unlike chronic ulcerative colitis. Mucosal ulceration, submucosal suppuration, thickening of all the coats of the wall, serosal infiltration, polyposis and cicatricial changes, especially in the submucosa, occur.

Symptomatology is varied; there is no typical syndrome: abdominal cramps, some weight loss, low grade fever are the most frequent symptoms, anemia is usually present: the sedimentation rate is an indication of the activity of the disease.

[1] Brown, L., and Sampson, H. L., Intestinal Tuberculosis, Lea & Febiger 1926.

Diagnosis is dependent upon roentgenological findings: a fuzzy, irregular bowel outline, narrowing, hyperirritability, shortening, and loss of haustration in a colonic segment, with a normal rectum. Proctoscopically the rectum and terminal sigmoid are normal, with few exceptions.

Primary resection and anastomosis and cecostomy; obstructive resection (Rankin) or a Mikulicz operation is now rarely done if medical measures fail. Even if successful, recurrences are the rule, thus Dixon and his associates at the Mayo Clinic emphasize that wide resection at an appropriate interval, is the treatment of choice.

Actinomycosis of the cecum, the usual site of this infection in the colon, is rare. Diagnosis is based on finding "sulphur bodies" in the discharge and demonstrating actinomyces on staining. If resection be required, a hemicolectomy and ileocolostomy is necessary but multiple fistulas and extensive disease may make the case inoperable. Adequate antibiotic, preferably neomycin or albamycin, therapy should be given before surgery is advised.

Lymphogranuloma usually involving the anorectum principally, may require rectal resection because of an almost complete rectal stricture and extensive perianal sinuses and an adjoining skin and subcutaneous infection. A perineal resection of the rectum after preliminary colostomy or an abdominoperineal resection can be done. Perirectal infection and fibrosis may make it more difficult than similar interventions for other lesions. Colostomy is not adequate, as a rule. Only the exceptional case of lymphogranuloma requires resection.

CHRONIC ULCERATIVE COLITIS

Chronic ulcerative colitis (idiopathic, nonspecific, colitis gravis) is an inflammatory disease of the colon and rectum characterized by multiple, minute, closely aggregated mucosal ulcers which bleed readily.

The disease begins in the rectum in 90 per cent of cases and progresses upward, extending along the lymphatics and perhaps by continuity in the mucosa; confluence of the petechial ulcers produce the large irregular "secondary" ulcers, destroying in some severe or protracted cases, large areas of the mucosa. As a result of the marked thickening and contraction of the submucosa, the bowel lumen and the length of the bowel is lessened; when extreme the "lead pipe" colon results. In 6 to 8 per cent only a part of the colon is affected: the segmental type. Remissions and exacerbations are characteristic, but the course may vary from the rapidly progressive fulminating, to the type limited to the rectum and having little tendency to progress. Healing does occur but much more often a granular contracted bowel remains even when the patient is clinically cured and having no symptoms. Arthritis is a rather frequent complication, anorectal abscess or fistula is not unusual and sinuses may involve adjacent structures *e.g.* the uterus, bladder or vagina. The symptoms are those of an infectious disease: malaise, exhaustion, fever, anemia, weight loss; and bowel involvement: anorexia, abdominal discomfort, gas, cramps, diarrhea with blood.

The diagnosis is made by proctoscopic examination, the extent and stage of the disease, by roentgenological means.

A granular mucosa which bleeds easily from minute ulcers, is generally seen proctoscopically (Bargen's streptococcic type). It often appears dry or glazed as mucus is wiped off and in more severe or advanced cases, it is granular. It may or

may not be reddened, but it usually is. The distribution of the characteristic hemorrhagic ulcers has been described as "pock-like" and the mucosa as granular, mucus is wiped off and in more severe or advanced cases, it is granular. It may or moth-eaten, bleeding (Buie[1]). Stippled oozing mucosa, after swabbing is characteristic.

The above mentioned irregular ulcers of a few millimeters to several centimeters may be found; the presence of the above described mucosa between them clarifies the diagnosis, differentiating them from the irregular ulcers sometimes seen in acute amebiasis and other diseases. In the latter the mucosa between the ulcers is not pitted, etc. This is an extremely brief and incomplete description of the proctoscopic findings; viz. Buie[1] for a more detailed statement. A granular, pitted, easily bleeding mucosa in a contracted bowel offers an easy diagnosis; the early changes and atypical findings offer diagnostic problems.

The treatment of chronic ulcerative colitis is medical. This consists of placing the patient on a high caloric, high vitamin, low residue diet and the removal of foci of infection, if any are found.

Medication is of definite but not infrequently of limited value; the diarrhea can usually be controlled by adequate doses of the deodorized or camphorated tincture of opium; bismuth, kaolin, etc. Because the bacterial flora of the colon were not considered susceptible to penicillin or the sulphonamides when these drugs were first produced, their use was not advocated, but capable clinicians using them in a number of cases since then, favor continued trial and insist they benefit some cases; hydrocortisone has its advocates. Intravenous solutions of dextrose, normal saline and whole blood are often required in the very acute phases of the disease.

Surgical intervention is for the purpose of establishing drainage proximal to the ulcerated bowel; this is accomplished by an ileostomy.

Two widely different schools of thought exist as to operative measures: the conservative, and the early ileostomy proponents. The former reserves ileostomy for the complications of the disease *e.g.* obstruction, internal fistulæ, pelvic infection with abscess, extensive anorectal suppurative infections, etc. and in a few uncomplicated cases which have not responded to careful prolonged medical management. The latter school of thought give the following indications for surgical intervention:

1. Intractibility on medical treatment with incapacity persisting for three months or more each year, 2. obstruction of the colon because of cicatricial change, 3. subacute perforation, sinuses, abscesses or fistulæ, 4. persisting hemorrhage, 5. infectious arthritis, 6. polypoid change, 7. carcinomatous change.

The practice of most capable surgeons today favors the conservative attitude with such modification as the case in hand dictates. If intractable under adequate medical management, colectomy is required; these comprise about 7 per cent of the cases reported by the Mayo Clinic; 20 per cent at the Leahy Clinic.

DIVERTICULITIS

Diverticulitis is an inflammation of diverticula of the large bowel. Diverticula may occur singly, or more often in numbers, throughout the whole bowel, but usually limited to the sigmoid segment. As a rule, they do not produce any symptoms, the patient not even being aware of their presence, save when inflammatory change takes place.

[1] Buie, L. A., Practical Proctology, Saunders, 1938

The pathogenesis of diverticulitis somewhat simulates an acute appendicitis, with one important difference: the diverticulum is short and its blood supply is not readily cut off. Hence, gangrene and perforation are slow in formation and the process becomes walled off before perforation takes place. In about one-third of diverticulitis cases a lower left quadrant mass may be felt, and in about one-fourth of cases visceral symptoms may predominate. Blood in the stool is not an unusual finding, however it is not of serious consequence ordinarily nor indicative of carcinomatous change. While carcinoma does occur with diverticulitis, nevertheless it is not a very frequent association. The differential diagnosis may be made by roentgenological examination which reveals the following:

Carcinoma	Diverticulitis
1. Filling defect with narrowing lumen having irregular borders	1. Filling defect may be pointed or cone-shaped.
2. Dilatation often present above the defect. Some dilatation below due to pressure of enema	2 Dilatation is less pronounced, except when there is an inflammatory or granulomatous mass co-existing.
3 Invagination of colon above the defect strongly suggestive of carcinoma	3. Rounded extra-luminal shadows of diverticula present; especially after evacuation
4. Area of involvement is shorter and the defect is more constant	4. Greater length of normal bowel is involved.

In about one-fifth of cases an exact diagnosis cannot be made preoperatively and at times resection of the mass may be necessary to arrive at the accurate diagnosis.

The treatment of diverticulitis is medical; surgical intervention has until recently been reserved for complications, as (1) perforations; (2) abscess formation; (3) inflammatory obstruction; (4) fistula, and (5) malignant changes. In general, operation should be delayed until the acute symptoms have subsided, or if it is apparent that they are dangerously progressing, or the abscess has become localized. The type of operation will depend upon the complication present. In most cases, a drainage operation, such as a temporary colostomy or cecostomy is indicated. A colostomy diverts the fecal current away from the lesion, thus with long continued drainage the inflammatory changes will often subside and the continuity of the lumen can be re-established later. Sometimes further surgery can be avoided.

Since the introduction of antibiotics, the mortality rate in the operative treatment for diverticulitis has been lowered from about 15 per cent to 5 per cent. Sulfasuxadine or sulfathaladine with or without penicillin were the chemotherapeutic agents chiefly used; now neomycin or achromycin is preferred. Until recently primary resection of the sigmoid in the face of an acute infection was considered a dangerous procedure. However, it is still a grave operation, especially if there is considerable associated inflammation in the tissues at the site of the diverticulitis.

Primary resection and anastomosis without preliminary colostomy may be done if there is no obstruction, if the inflammation is not severe, or when only a small segment of the bowel is involved. A preliminary colostomy is the procedure of choice in all cases of obstruction or perforation; if indicated, resection may be done later. In a very few instances, when there is little inflammatory reaction in the tissues, the local excision of a single inflamed sac or even a rectosigmoidal fistula may be done and the bowel opening sutured. It is important to construct the colostomy so that fecal diversion is complete. This results in a diminution of symptoms

even in sigmoidovesical fistula. The latter usually requires first a colostomy, then relief of urinary infection and at a later date separating the fistula from the bladder and resection of a segment of the bowel. An attempt at repair in one stage invites *failure or disaster, in other than the exceptional case.*

BENIGN TUMORS

Adenomas comprise the vast majority of benign tumors of the colon, and are of added importance because of their tendency to become malignant; the term polyp, still much used, is generic, not specific. Papillomas are much less frequent. "There is probably no tissue, (other than colonic adenoma) which illustrates so strikingly the transition of an inflammatory over-growth into a malignant tumor" Ewing. "Papillomata of the bowel can certainly be regarded as pre-cancerous lesions" Boyd.

Other benign tumors of the colon are lipoma, leimyoma, fibromyoma, and angioma; these, except lipoma, are infrequent; rarely melanoma, carcinoid and others are reported. Neoplasms, primary elsewhere *e.g.* sacrococcygeal chordoma, endometrioma, carcinoma of the prostate, sarcoma, etc. which may involve or invade the bowel, are not considered here. Adenomas of the rectum and sigmoid colon are discussed later.

Single adenomas from 15 cm. above the anus, or thereabout, if producing symptoms, *e.g.* bleeding, diarrhea, pain, etc., require excision. This is generally best done by colotomy, clamping the base including a little of the mucosa, excising the adenoma over the clamp, taking a few under and over stitches of a continuous suture through the stump. (Fig. 621) A longitudinal incision is made midway between the two tænia coli on the antimesenteric border; it is usually done here as it is the most accessible site. Rankin advocated this as a result of his and Stewart's study of the vascular structure of the colon wall.

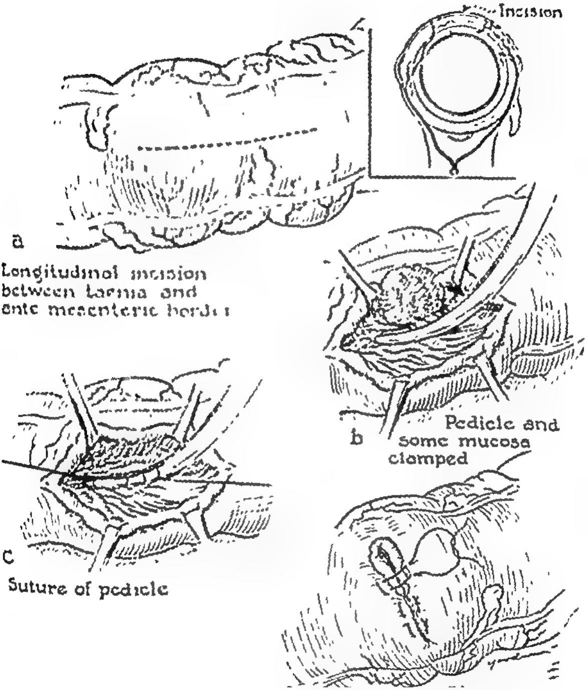
Sessile adenomas of 2.5 cm. or more in diameter, or a localized area of adenomatosis of such size may be removed as the single adenoma above, but ordinarily require a resection of the colon for adequate removal; a "wedge" resection if of about 2.5 cm, diameter. resection of the necessary amount of the colon if larger.

Papillomas (papillomatous adenoma, villous tumor) are not rare, but are not nearly as frequent as ordinary adenomas. They deserve consideration as a particular group, because in them it is especially easy to overlook an area of carinoma in the base, or pedicle if pedunculated. They do not have a pedicle as a rule, if they do, it is apt to be broad. They are usually not small when seen at operation so resection of the bowel is necessary for adequate removal, but particularly for later histological study of the whole tumor.

Polyposis (multiple polyposis) may be congenital or the aftermath of extensive ulceration. The polyp studded colon of a healing or healed chronic ulcerative colitis or a tuberculous enterocolitis evidence the latter type. This has been called "false polyposis" as most of the projecting knobs and tags are not true adenomas. Although hemicolectomy may at times be performed in chronic ulcerative colitis, polyposis is not the primary reason, and in the tuberculous the patient is practically never operable, because of the pulmonary disease.

The congenital variety is of chief surgical importance. A large percentage, probably the majority, of these patients die of carcinoma. Experience has shown that the procedure of choice is either a colectomy in one or two stages and a perma-

nent ileostomy; or a primary ileostomy, then a colectomy in one or two stages and a subsequent ileosigmoidostomy, the ileostomy being closed later. The ileosigmoidostomy can be done, of course, only if the rectum is free or can be freed of polyps by fulguration. Multiple operations of this sort are ordinarily safe only in the hands



CHAPTER 74

DISEASES OF THE LARGE BOWEL—DOLICHOCOLON AND MEGACOLON

By John L. Keeley

Disturbances in colonic function may be associated with increased length, dolichocolon, or, more commonly, both increased length and caliber, megacolon. The extensive surface area of mucosa in such abnormalities results in greater absorption of water. Fecal material then becomes firm and relatively dry, resists the propulsive effect of peristalsis and constipation results. Stercoraceous ulceration may occur but rarely causes perforation of the bowel. The redundancy of the colon and the weight of its contents may lead to volvulus.

DOLICHOCOLON

Dolichocolon is the term applied to a congenitally long colon. It is rare. The bowel wall is intrinsically normal. However, the large surface area absorbs so much water that inspissated fecal masses form and constipation develops. Chronic constipation may be the chief complaint in some patients while others may have normal bowel movements between acute attacks of obstruction due to recurrent volvulus of the sigmoid.

Some patients may have symptoms from birth distinguishing dolichocolon from megacolon due to chronic constipation. Nevertheless in many patients, the first sign of dolichocolon is the occurrence of sigmoid volvulus in adult life.

It is important to distinguish dolichocolon from the various types of megacolon to be described below. Medical management can be helpful in most patients with dolichocolon, but it is important to remember that when medical management is ineffective or in patients in whom recurrent volvulus occurs resection of the redundant sigmoid is the best treatment. Resection of the sigmoid decreases the area for absorption permitting a higher water content in the stools, which is a significant factor in correcting constipation. In addition, volvulus can not occur when redundancy has been eradicated.

Because dolichocolon is an uncommon lesion and because it can often be treated medically, surgery will be indicated in a small group of patients with this disturbance. For this small group anterior rectosigmoid resection is the procedure of choice.

Emphasis should be placed on the preparation of the bowel for surgery. This includes mechanical cleansing by enemas, the use of cathartics, and the new group of preparations such as Colace and Doxinate. The bacterial flora, particularly the coliform organisms, is decreased by sulfasuxidine for three to four days and neomycin for the final twenty-four hours of the preparation period.

General anesthesia is preferred. The abdomen is opened through a long left rectus muscle displacing incision. The exposure of the colon and rectum in the

pelvis is facilitated by emptying the bladder, and Trendelenburg's position aids in displacing the small bowel into the upper abdomen. The peritoneum on either side of the mesosigmoid is incised and the branches of the sigmoid and left colic arteries supplying the section of colon to be removed are ligated and divided. Wide

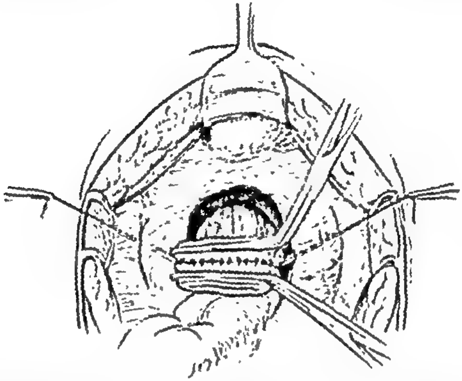


FIG. 622.—Anterior resection of the colon. The redundant portion has been excised. The posterior row of interrupted silk sutures has been placed and tied. The two most lateral sutures are used for traction and guides. The bowel ends held in the clamps are excised, removing all crushed tissue.

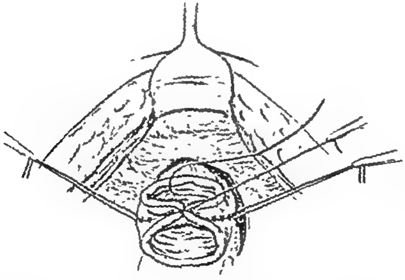


FIG. 623.—Interrupted chromic catgut sutures approximate all layers of the bowel segments and form the inner suture line.

excision is not necessary as in malignant disease, therefore, the superior hemorrhoidal artery can be preserved. The dissection is carried distally only enough to provide a peritoneal cuff which can be attached above the level of the completed anastomosis which then occupies an extraperitoneal position.

The details of the anastomosis are as follows: an outer posterior row of intestinal silk sutures is placed first; the lateral ones are used for traction (Fig. 622). Interrupted fine chromic catgut sutures through all layers of the bowel complete the posterior half of the anastomosis (Fig. 623). A similar layer is placed in the anterior half of the anastomosis which is finally completed by a row of interrupted silk sutures. The edges of the peritoneum are then attached to the bowel wall placing the anastomosis extraperitoneally (Fig. 624). The abdomen is closed without drainage.

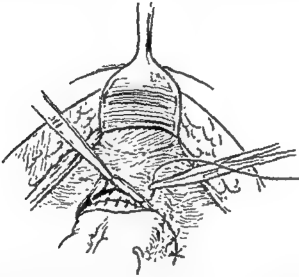


FIG. 624.—The anterior half of the anastomosis is completed by a row of interrupted catgut sutures which invert the edges of the bowel and a row of silk sutures in the seromuscular layer. The flap of peritoneum is then "tacked" to the serosa of the bowel above the anastomosis.

MEGACOLON

Megacolon can be divided into three groups: organic, functional, and aganglionic megacolon. The therapeutic management depends on the type and, therefore, it is important to arrive at an accurate diagnosis of the underlying etiologic factor.

A. *Organic megacolon* is the name given to those lesions which have a grossly demonstrable cause. Thus, pelvic tumors causing extrinsic pressure, stenosis following imperforate anus repair, or, rarely, congenital stenosis of the rectum or rectosigmoid may cause megacolon. Obviously the megacolon is a *secondary* effect and correction of the pathology causing it is indicated.

B. *Functional Megacolon.*—In this type there is a history of constipation during infancy but if accurate information is available it is clear that the onset was not in the first days of life. Formula and diet may be important factors and eventually fecal impaction, fissure and sphincter spasm form a vicious circle which prompts the child to resist all attempts at toilet training. The increase in length and diameter is greatest in the sigmoid and rectum, probably due to the nature of their contents, the factor of stasis and the looser attachment of the sigmoid compared to that of the descending colon

The problem becomes worse when the soft stool of infancy is replaced by the firmer stool of childhood or adult life. Abdominal distension of significant degree does not occur in functional megacolon but a fecal filled sigmoid may be palpable. The rectal sphincters are normal. Fecal impaction is encountered immediately above them. Impaction leads to occasional incontinence of feces. Barium studies

show the rectal ampulla dilated to the anal canal (Fig. 625). Post evacuation films usually show poor emptying.

Medical management almost always will suffice in the cases of functional megacolon. In a rare instance, after failure of prolonged and faithful medical management, intra-abdominal resection of the redundant sigmoid as described for dolichocolon may be indicated to decrease the area of absorption and to prevent volvulus.

C. *Aganglionic Megacolon (Hirschsprung's Disease).* The many earlier etiologic explanations for megacolon have been brushed aside by the establishment of this type as a particular entity in which the pathology is in a short "inactive" segment usually limited to the rectum. Hirschsprung's original description, while emphasizing the large caliber and extra length of the colon, did include the observation that the rectum "seemed to be the site of some kind of narrowing."

FIG. 625 Barium enema in a patient with megacolon of the functional type. The colon is increased in both caliber and length right down to the level of the sphincters.

Among some of the earlier explanations was "autonomic imbalance." However, careful studies by Whitehouse and Kernohan, Dalla Valle, Cameron, and by Bodian, Stephens and Ward have shown that the fundamental pathology is the congenital absence of ganglion cells in Auerbach's plexus in the region of the internal sphincter, rectum, and rectosigmoid and in rare instances even the entire colon.

Hiatt and Swenson, Rheinlander and Diamond have shown that although contractions occur in these aganglionic segments they are not organized effective peristaltic movements but mass or tonic contractions instead. The fecal masses are propelled by the normally innervated colon to the level of the inactive segment and progress is arrested there. This stasis leads to dilatation and elongation of the colon proximally. Hypertrophy of its walls develops in response to the obstruction.

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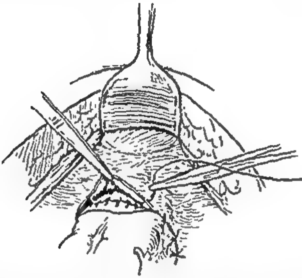

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Megacolon can be divided into three groups: organic, functional, and aganglionic megacolon. The therapeutic management depends on the type and, therefore, it is important to arrive at an accurate diagnosis of the underlying etiologic factor.

A. *Organic megacolon* is the name given to those lesions which have a grossly demonstrable cause. Thus, pelvic tumors causing extrinsic pressure, stenosis following imperforate anus repair, or, rarely, congenital stenosis of the rectum or rectosigmoid may cause megacolon. Obviously the megacolon is a secondary effect and correction of the pathology causing it is indicated.

B. *Functional Megacolon.*—In this type there is a history of constipation during infancy but if accurate information is available it is clear that the onset was not in the first days of life. Formula and diet may be important factors and eventually fecal impaction, fissure and sphincter spasm form a vicious circle which prompts the child to resist all attempts at toilet training. The increase in length and diameter is greatest in the sigmoid and rectum, probably due to the nature of their contents, the factor of stasis and the looser attachment of the sigmoid compared to that of the descending colon.

The problem becomes worse when the soft stool of infancy is replaced by the firmer stool of childhood or adult life. Abdominal distension of significant degree does not occur in functional megacolon but a fecal filled sigmoid may be palpable. The rectal sphincters are normal. Fecal impaction is encountered immediately above them. Impaction leads to occasional incontinence of feces. Barium studies

show the rectal ampulla dilated to the anal canal (Fig. 625). Post evacuation films usually show poor emptying.

Medical management almost always will suffice in the cases of functional megacolon. In a rare instance, after failure of prolonged and faithful medical management, intra-abdominal resection of the redundant sigmoid as described for dolichocolon may be indicated to decrease the area of absorption and to prevent volvulus.

C. *Aganglionic Megacolon (Hirschsprung's Disease)*. - The many earlier etiologic explanations for megacolon have been brushed aside by the establishment of this type as a particular entity in which the pathology is in a short "inactive" segment usually limited to the rectum. Hirschsprung's original description, while emphasizing the large caliber and extra length of the colon, did include the observation that the rectum "seemed to be the site of some kind of narrowing."

FIG. 625 -Barium enema in a patient with megacolon of the functional type. The colon is increased in both caliber and length right down to the level of the sphincters.

Among some of the earlier explanations was "autonomic imbalance." However, careful studies by Whitehouse and Kernohan, Dalla Valle, Cameron, and by Bodian, Stephens and Ward have shown that the fundamental pathology is the congenital absence of ganglion cells in Auerbach's plexus in the region of the internal sphincter, rectum, and rectosigmoid and in rare instances even the entire colon.

Hiatt and Swenson, Rheinlander and Diamond have shown that although contractions occur in these aganglionic segments they are not organized effective peristaltic movements but mass or tonic contractions instead. The fecal masses are propelled by the normally innervated colon to the level of the inactive segment and progress is arrested there. This stasis leads to dilatation and elongation of the colon proximally. Hypertrophy of its walls develops in response to the obstruction.

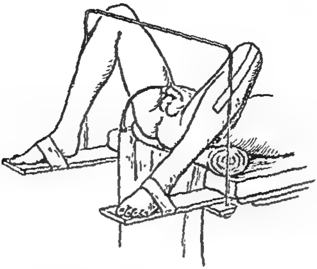


FIG. 627.—Position of patient for simultaneous exposure of abdomen and perineum. The urethral catheter keeps the bladder empty during the operation and in the postoperative period. *It should not be thought of as a guide to protect the urethra as the dissection should at no time stray even slightly from the bowel wall.*

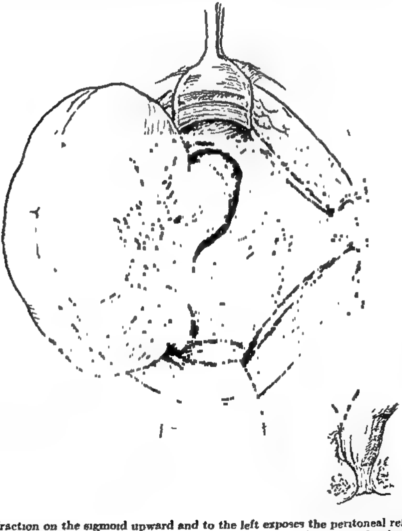


FIG 628 —Traction on the sigmoid upward and to the left exposes the peritoneal reflection which is divided as shown here The insert emphasizes the manner of freeing the rectum by dividing all attachments at the point where they join the rectal wall.

for the adequate dissection of the rectum deep in the pelvis. Successive abdominal and perineal exposure as used in the standard abdomino-perineal resection of the rectum is quite satisfactory. The "pull-through" portion of the perineal stage can be accomplished beneath standard laparotomy drapes and the position and tension of the colon in the pelvis established before closing the abdominal incision.

The abdomen is opened through a long left rectus incision beginning at the level of the symphysis and displacing the muscle laterally. The small bowel is packed away and a biopsy of the muscle layer of the colon is done at the upper level of proposed resection to insure excising all of the aganglionic segment. This can be done without opening the mucosa. While awaiting the report, the peritoneum on either side of the mesosigmoid and across the cul de sac may be incised (Fig. 628).

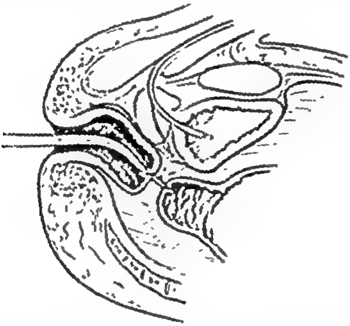
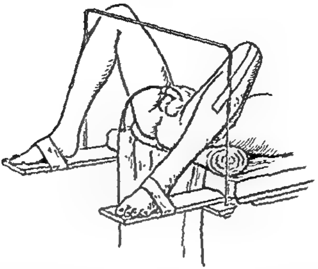


Fig. 629.—The rectum has been freed down to the anal sphincters. A segment of redundant bowel has been removed and the rectal and colonic ends closed by sutures which are then tied together.

Dissection of the rectum is done with fine, sharp scissors. All vessels and strands of connective tissue are divided at their point of attachment to the rectal wall thus avoiding damage to the nerve supply of the anal and bladder sphincters and to the ejaculatory apparatus in males. The dissection is carried down to the levator muscles (Fig. 628). The sigmoid and rectum are freed by dividing the mesosigmoid and superior and middle rectal (hemorrhoidal) vessels.

The blood supply to the hypertrophied bowel is usually adequate as the vessels are larger and appear to be more numerous than usual. The descending colon may be mobilized and the left colic artery may be divided if necessary to bring the colon down for the anastomosis. Extremely careful hemostasis is necessary as the cul de sac must be dry to guard against pelvic hematoma or abscess. The dissection deep in the pelvis may be facilitated by the excision of the bulky portion of the hypertrophied bowel. The ends are then closed with a Von Petz clamp or sutures (Fig. 629). This specimen can then be studied for the presence of ganglion cells. Removal of this bulky segment in infants and children avoids the problem of drawing it through a small anal opening.

A curved clamp is inserted into the rectal segment and grasps the end of the closed rectal stump. With the closed bowel ends attached by sutures, traction on



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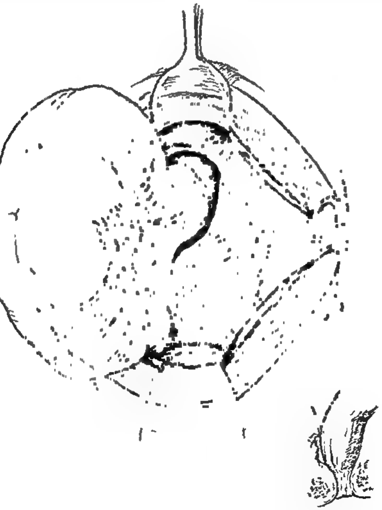

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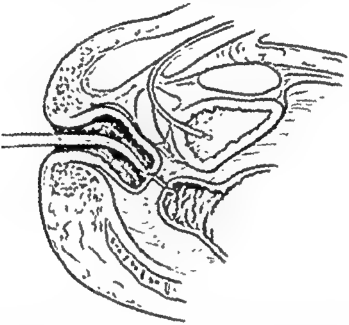

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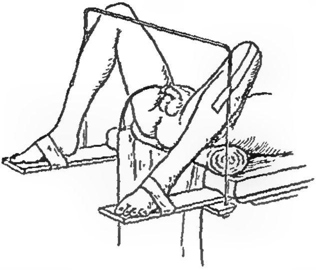

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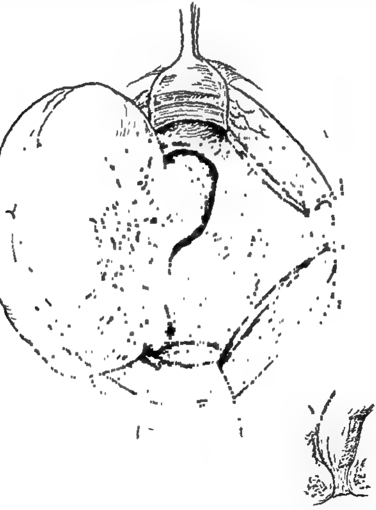

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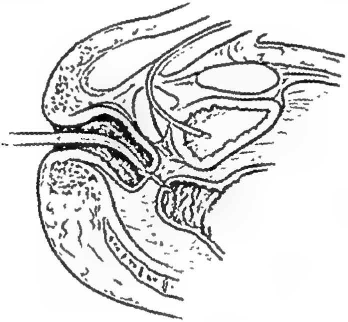

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A curved clamp is inserted into the rectal segment and grasps the end of the closed rectal stump. With the closed b[illegible] attached by sutures, traction on

the forceps turns the lower segment inside out and draws the upper segment into the lower one producing a relationship between the two similar to that seen in rectal prolapse of severe degree (Fig. 630).

Hiatt does not interrupt the continuity of the bowel but simply turns it inside out by reaching up the rectum with forceps and producing what is essentially a large rectal prolapse (Fig. 631). If this procedure is followed, it is important that

Fig. 630.—Traction on the rectal stump from below turns it inside out and "intussuscepts" the upper segment into the lower one. Both bowel segments then protrude through the anal canal and the protruding mass closely resembles that seen in a large rectal prolapse.

Fig 631 —With the bowel freed all the way down to the region of the sphincters it can be turned inside out by traction as shown The relation of the walls of the rectal and colonic segments is then similar to that shown in Figure 630

the weight of the prolapse be prevented from pulling down too much bowel to be excised. Should this occur, the anastomosis will be under tension which will increase when the taut bowel is distended with gas or feces in the postoperative period. Should the bowel preparation be found inadequate at operation, fecal masses can be "milked" out of the bowel into and through the rectal prolapse. Contamination of the peritoneal cavity is avoided by this maneuver and no collection of feces is allowed to remain above the anastomotic site.

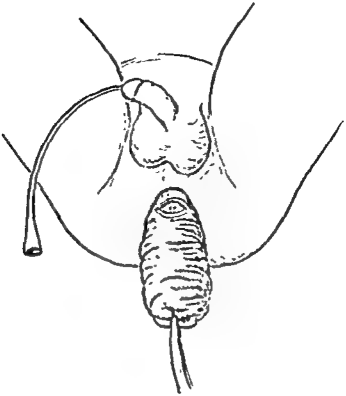

Fig. 632.—An incision is made through the wall of the rectum 1.5 to 2 cm. from the pectinate line and the serous surface of the colon segment is exposed

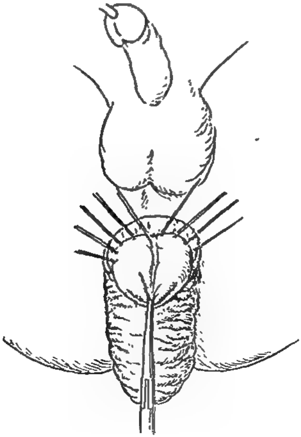

Fig. 633.—A row of silk sutures approximates the surfaces of the rectal stump and the colon segment which it encircles. The posterior half of this row of sutures is completed as the amputation of the ensheathing rectal stump is accomplished

Whether a segment of bowel is removed (as in the Swenson procedure) or not (as practiced by Hiatt) the "pull-through" maneuver will result in the protrusion shown in Figure 632.

The perineal field is thoroughly cleansed, prepared and redraped. The amputation of the protruding bowel and the subsequent anastomosis is shown in Figures 632, 633, and 634.

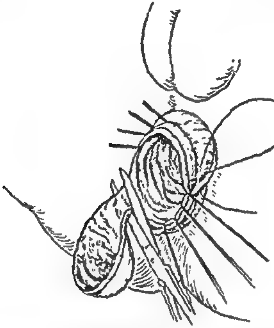

FIG. 634.—The protruding colon is amputated at the same level as the everted rectal stump. A row of interrupted chromic catgut sutures through all layers completes the anastomosis

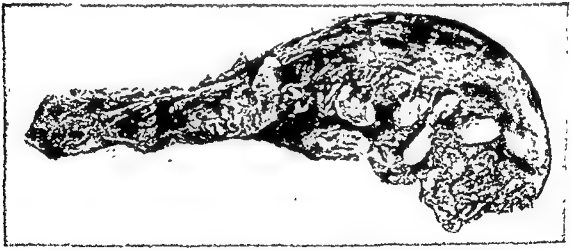

FIG. 635.—Specimen of rectosigmoid resected in patient with Hirschsprung's disease. The lower small caliber inactive segment is in contrast to the dilated and hypertrophied portion of the sigmoid.

A circular incision $1\frac{1}{2}$ to 2 cm. from the pectinate line is made through the outer layer of the prolapse (Fig. 632). The upper edge of this incision is turned back and a row of seromuscular sutures of silk is begun as shown in Figure 633. It is strongly recommended that the "cut and sew" technique be followed here. This is done by continuing the circular incision of the outer layer a segment at a time and installing the seromuscular sutures as the serous surfaces are exposed. The seromuscular sutures may be completed before the inner bowel segment is incised.

Here again the "cut and sew" technique is followed; the interrupted sutures of fine chromic catgut can be put in place as the amputation of the protruding bowel progresses (Fig. 634).

When completed, the anastomosis, which is of the end-to-end variety is pushed through the sphincters. A cul de sac drain is effective in preventing a collection of serosanguinous fluid and can be inserted through a stab wound in the mid-line just anterior to the tip of the coccyx. Figure 635 shows a typical specimen. Good results may be expected in over 90 per cent of properly selected patients treated by adequate operation.

The complications of rectosigmoidectomy by either the Swenson or Hiatt procedures are infection, either peritonitis or pelvic abscess, urinary retention which is transitory, anastomotic stricture or fistula and recurrence due to incomplete removal of the aganglionic segment. There is insufficient information on which to base conclusions concerning ultimate ejaculatory function but Swenson reports the cases of four adults in whom this function was not disturbed. The mortality depends on the age and initial general condition of the patient and the avoidance of operative and postoperative complications. It should not exceed 5 per cent and is lower than this in some reports.

QUESTIONNAIRE

1. What are the three types of megacolon? Contrast the history and findings in patients with each type. How does dolichocolon differ from each type of megacolon?
2. What are the indications for bowel resection in dolichocolon and functional megacolon? Name two objectives achieved by this operation.
3. What manifestations of Hirschsprung's disease may be present in the newborn?
4. What is the etiology of Hirschsprung's disease?
5. What is the purpose of recto-sigmoid resection in its treatment?
6. What are the indications for colostomy in Hirschsprung's disease?
7. Describe the technique of rectal biopsy to establish the diagnosis of Hirschsprung's disease. In which patients is this found to be most useful?
8. What is the most important technical point in freeing the rectum in the pelvis? If done properly what complications is it designed to prevent?
9. What are the postoperative complications of rectosigmoid resection and how can they be avoided?
10. What is a reasonable mortality rate for rectosigmoidectomy?

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of a portion of the vaginal wall adds no great risk but a hysterectomy usually does. Fixation is more often a contraindication in men than in women. In general, if extrarectal infiltration is palpable, fixing the organ, the lesion is inoperable, or *questionably so.* *This is determined preoperatively. Borderline* cases are determined at laparotomy. If the neoplasm is above the reach of the finger, some information can often be had by manipulating it with the proctoscope. However, operability in cases not palpable by digital examination is ordinarily decided at laparotomy; whether it is advisable or even justified by the presence of metatases, *e.g.* mesenteric, para-aortic, hepatic, etc.

Lymphatic Spread in Cancer of Large Bowel.—At laparotomy the carcinoma commonly is seen as a 5 to 8 cm. long mass in the colon, often with a depressed or puckered area on the serosal surface. As it spreads locally it reaches the serosa, *where neoplastic and inflammatory changes produce adherence to adjacent structures,* and eventually fixation to them. The liver, gall bladder, stomach, pancreas or spleen are among the organs most often involved. The small intestine, or abdominal wall may be involved; the lateral peritoneal sulcus in cecal lesions. Fixation is most common in the rectum and sigmoid; in males to the posterior wall or base of the bladder, less often to the prostate, in females, to the posterior vaginal wall, cervix or uterus.

Metastases usually spread in an orderly step by step extension from the primary site along the lymphatic course, thus the area of probable lymphatic involvement must be clearly understood. The size of a node is not a criterion of malignancy, although it is suggestive if it is large, induration infers carcinomatous invasion but it may well be only inflammatory. The size of the tumor itself is not a dependable criterion of metastases, these may be present with a small neoplasm and absent with a larger one. This is the chief reason why it is a recognized surgical principle that "small operations are not done for small cancers" and *larger ones for large* cancers. (Jones, T. E.). If retrograde (afferent) lymphatic extension occurs it indicates the efferent channels are invaded and blocked. Eliminating the area of lymphatic extension is essential. The two factors of chief importance in prognosis are: whether lymphatic extension is present or absent and the degree of malignancy.

A few doubtful or small hepatic nodules, (old tubercles, bile-duct adenomas, lymphangiomas), etc. do not necessarily contraindicate resection, but there well may be deeper metastases not palpable; palliative resections, imposing the grave risk of an extensive operation upon a person having cancer in the liver, require the best judgment of an experienced surgeon.

It is a basic principle that any operation for cancer of the colon must be radical enough to remove the growth together with as much of the lymphatic tissue along the ordinary course of extension as may be safely done; the accompanying diagrams illustrate this.

Cancer of the Cecum and Ascending Colon.—*The plan of operation* for cancer of the cecum or the ascending colon, including removal of the cecum, ascending colon, and lymphatic glands along the right colic artery is shown in Figure 636. The hepatic flexure is removed to eliminate sufficient lymphatic area and to facilitate the ileo-colostomy subsequent to resection and to assure adequate circulation for the anastomosis.

Cancer of the Hepatic Flexure.—When the hepatic flexure is involved the extent of resection includes the removal of the entire right colon, plus the right half of the of the transverse colon. In this case, the following vessels are ligated: ileocolic,

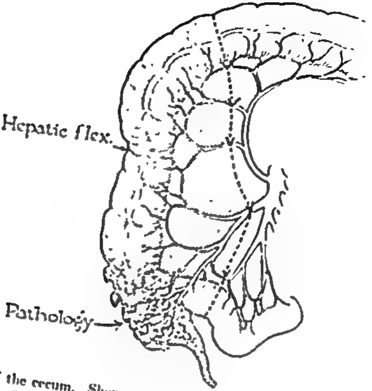


FIG. 636.—Carcinoma of the cecum. Shows extent of excision in order to remove all possible sources of spread through the lymphatics.

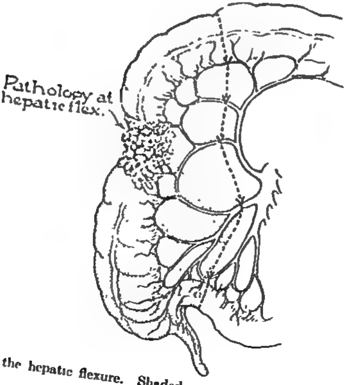


FIG 637.—Carcinoma of the hepatic flexure. Shaded area represents the extent of excision.

right colic, and middle colic. The ligation of these vessels makes it possible to remove with the growth the lymph glands draining the hepatic flexure.

Cancer of the Transverse Colon.—The lymph channels draining the transverse colon follow along the middle colic artery, hence new growths in this area require

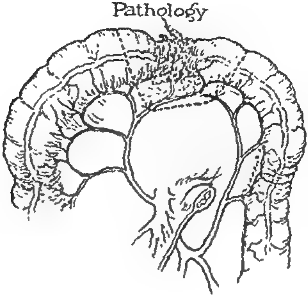


Fig. 638.—Extent of excision for carcinoma of the transverse colon

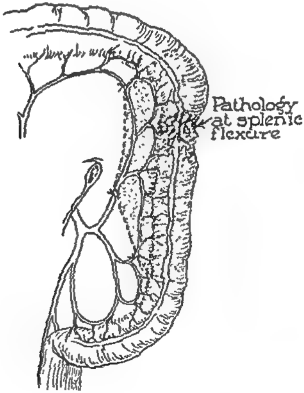


Fig. 639.—Shows the extent of resection for carcinoma of the splenic flexure.

ligation of the middle colic artery and the anastomotic branch of the left colic. In most cases the middle colic artery is long enough so that it is possible to remove the tumor without endangering the blood supply (Fig. 638).

Cancer of the Splenic Flexure.—The lymphatics draining this part of the bowel follow the course of the left colic artery, hence this artery must be ligated. Ligation

of the left colic artery necessitates the excision of the colon from the junction of the middle or the left third of the transverse colon to the middle of the descending colon (Fig. 639).

Cancer of the Descending Colon.—When excising a carcinomatous growth involving the descending colon, the left colic and the upper sigmoidal arteries are ligated. This necessitates the removal of the splenic flexure, if the growth is high, and the upper half of the pelvic colon, when low (Fig. 640).

Cancer of the Pelvic Colon.—The lymph glands of this part of the colon follow the course of the sigmoidal, superior hemorrhoidal and inferior mesenteric arteries. In the above case (Fig. 641) the upper limit of resection is at the middle of the de-

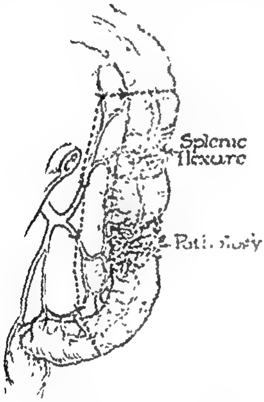


FIG. 640

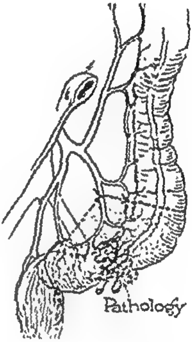


FIG. 641.

FIG. 640.—Extent of resection for carcinoma of the descending colon.

FIG. 641.—Excision for tumor just proximal to the sigmoid, as commonly done; higher extension of the dissection and resection is now often used.

scending colon where the left colic artery enters; the lower limit is at the rectosigmoidal junction. More extensive resections, including one-half or more of the descending colon, are being increasingly performed.

Sudeck's point, as illustrated in Figure 642 can be disregarded in excision of the sigmoid and upper rectum, except in the Hochnegg "pull through" type of operation, *e.g.* the Babcock-Bacon modification.

Dixon states that the superior hemorrhoidal vessels may be sacrificed without impairment of the circulation to the rectum or rectosigmoid, as a result of his experience in over 200 cases in the last seventeen years; and Wangensteen reporting sixty-one operations concludes that "Sudeck's critical point has no importance in excision of the pelvic colon and the upper rectum. Probably only in the "pull through" operation is Sudeck's point really important."

In excision of a segment of the pelvic colon and its triangle of mesentery, it is often unnecessary to ligate the superior hemorrhoidal artery. In the case illustrated in Figure 641, ligation and excision is performed as indicated. If lymphatic involvement extends more toward the last sigmoid artery (sigmoidea-ima) the apex of the mesenteric triangle would be close to this artery, which would be clamped, cut and ligated; if the superior hemorrhoidal artery is implicated by metastases or in the area of probable lymphatic extension, it can be sacrificed. In any event, in carcinoma of the pelvic colon, only the vessels to the involved triangle are ligated. The mesentery and its vessels beyond are carefully handled to avoid injury to them.

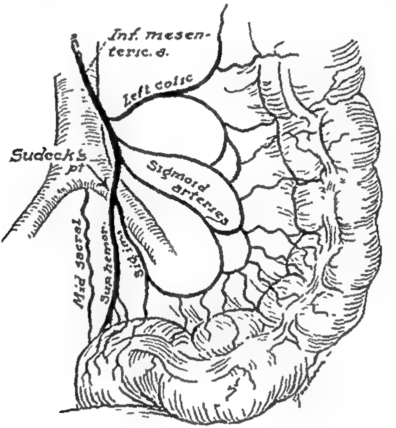


FIG. 642.—Critical point of Sudeck.

The junction between the lowest sigmoidal and the superior hemorrhoidal artery is called the critical point of Sudeck; space does not permit its further discussion here.

Preoperative Preparation.—*Hospitalization* of 2 to 5 days is required. A saline laxative, *e.g.* saturated solution of sodium phosphate twice daily to maintain liquid or semi-liquid bowel movements is given and warm water enemas twice a day. Drastic catharsis is not necessary; feces of a consistency readily removed by enema is the requirement. A low residue, high carbohydrate diet is given. Even with partial obstruction the above can be done. In more complete obstruction, intranasal gastric suction or aspiration by Miller-Abbot tube and hot abdominal compresses may relieve it. A saline laxative, administered by the tube, subsequently temporarily clamped off, may be cautiously used. One or two quart enemas are given twice daily until the return flow is clear. Colonic decompression cannot be obtained by aspiration; Miller-Abbot or Wangensteen suction is not effective in these cases. Decompression cannot be accomplished across the ileocecal valve.

Intravenous fluids are required to replace those removed by suction; this does not supply all the electrolytes thus removed; operation is indicated as soon as it is apparent that these measures of medical management are proving ineffective.

Preoperative Procedures.—The following preoperative preparation or some modification of it is commonly used, except in acute obstruction. These are notes for the resident in surgery.

1. The patient is to be in the hospital 2 to 5 days before operation.
2. Examine heart and lungs especially, order a chest roentgenogram (flat-plate, to determine pulmonary metastases, etc.) order E.K.G. if necessary.
3. Complete blood count, clotting and prothrombin time and serum protein determination.
   Type and cross match blood, Rh factor. If blood below 4,000,000, hemoglobin 70 per cent or serum protein 6 or less, give preoperative transfusion. Have 500 cc. of blood available in operating room and 500 cc. of plasma ready for immediate use.
4. High carbohydrate, high vitamin, non-residue diet; liquid diet the day before operation. Liberal fluids by mouth.
5. Urinalysis and total N.P.N.
   Catheterize and determine if residual urine present.
6. S.S. enema on day of entry, followed by hot tap water enema twice daily, as directed.
   Saline laxative in graded doses on entry if necessary.
7. Usually neomycin 0.5 g. 1st day 2 tab. q 6 hr., then 1 tab. q 6 hr.
8. Penicillin or Albamycin as indicated.
   Continue postoperatively as directed.
9. Seconal gr. 1½ at hour of sleep as required
   Seconal gr. 1½ the night before the operation.
   Nembutal gr. 1½, one and one-half hours preoperatively
   $\left.\begin{array}{l}\text{Morphine sulphate gr. 1/6 to 1/4}\\\text{Hyoscine hydrobromide gr. 1/150 to 1/200}\end{array}\right\}$ ¾ hour preoperatively.

CHAPTER 76

SURGERY OF THE LARGE BOWEL—TECHNIQUE OF OPERATIVE PROCEDURES

By Clement L. Martin

COLOSTOMY

By colostomy is meant the establishment of an artificial opening between the colon and the skin.

In acute obstruction of the colon the first and immediate consideration is to relieve the patient from the effects of obstruction by decompressing and draining the proximal bowel. If carcinoma, it can usually be excised at a later date when the patient is in better condition to withstand a more formidable operation.

In chronic obstruction with a thick walled and dilated colon a preliminary colostomy places the involved segment at physiological rest, thus making a subsequent radical resection less hazardous and protects the anastomosis by acting as a safety valve against the back pressure from gas. It is the only possible surgical procedure for inoperable carcinoma of the rectum or when a radical rectal resection is contraindicated.

In cancer, the colostomy is permanent, unless it is simply a step in a Mikulicz type operation; it may be temporary in diverticulitis or granulomas of the colon, either because a resection may be done later and the diseased tissue extirpated, or because the inflammatory mass gradually diminishes or disappears over a period of time.

There are two main types: the loop or double barrel variety and the single loop or end colostomy. The former has a much wider application, the latter is an integral part of abdominoperineal resection of the pelvic colon and rectum.

In carcinoma of the left half of the large bowel and in diverticulitis of the sigmoid or descending colon the operation can effectively be performed in the transverse colon to avoid appendicostomy or cecostomy with their distinct disadvantages (incomplete fecal diversion, small appendix in adults which will not admit large catheter, retrocecal appendix, etc.).

Colostomy of the ascending colon can be done but mobilization is more difficult and one may have to mobilize the hepatic flexure It is generally agreed that the sigmoid and transverse colon are the preferable sites and satisfy the requirements of most cases; if the obstruction is in the ascending colon or at the hepatic flexure the choice is between a cecostomy and a colostomy, the former generally preferable.

Ordinarily not difficult, the operation may be so because of obesity, a short mesosigmoid, a large tumor and extensive infiltration or adhesions, and in the acute case, marked distention. In immobile sections of the colon and often to increase mobility in the sigmoid or other mobile sections an incision is made in the lateral peritoneum through the "white line," if discernable, a thickening of the peritoneum

which marks its junction with the mesentery. It is avascular and can be incised without bleeding throughout its course. Blunt dissection of retroperitoneal fat is carried out with the fingers as traction is made on the colon and the loop of bowel gradually delivered.

With complete obstruction and great distention of the bowel, suturing the colon to the parietal peritoneum and leaving the opening in the depth of the wound may rarely be necessary. It does not divert the fecal stream and leaves an open, slowly healing infected wound. It generally confesses an inadequate surgeon.

Aspiration of the bowel with a needle and syringe, or continuous suction may aid mobilization when the colon is much distended, or used to relieve distention after the loop is brought out. A catheter can be inserted in the loop, held by a purse-string suture at operation. This is not often necessary, except in acute obstruction.

It is of primary importance to mobilize the bowel sufficiently so that it lies, without great tension on the mesentery and consequently on its vessels, well above the skin. Incising the lateral peritoneum assures this. A McBurney incision, generally on the left, is best as the tissues of the abdominal wall fit closely about the bowel and lessens the possibility of wound infection and later hernia.

Site of the Colostomy.—The site of a colostomy will depend upon the location of the lesion. Because of the accessibility of the sigmoid loop, and the ease of caring for it on the part of the patient, an inguinal colostomy is generally done for carcinoma of the rectum, although a left rectus site is also satisfactory. The colostomy may also be made in the transverse colon and the cecum according to the location of the growth. For carcinoma of the rectum the sigmoid has been employed more often for the opening than any other portion of the large bowel as it is mobile and the feces are usually solid or semisolid in this part of the bowel. As a rule, control after colostomy is much more satisfactory when the patient is slightly constipated, the stools being of formed consistency. If, as is often the case, the colostomy is a preliminary procedure to a subsequent excision of the rectum, the opening must be made sufficiently high on the sigmoid to permit subsequent wide excision of the lymphatics. The artificial anus can be made high up on the sigmoid colon by feeding the proximal slack back into the abdomen until a taut point is reached. Before performing a colostomy a thorough exploration of the abdomen is made, except in marked obstruction, to determine operability, lymphatic involvement, the presence or absence of metastasis, and the condition of the growth. The exploratory incision can be made in the mid-line or through the rectus muscle and a second incision in the inguinal region can be made for the colostomy, or the colostomy stoma left in the midline or paramedian wound.

Technique.—For more detailed description the inguinal type of colostomy will be described; see illustration: colostomy (Fig. 643).

A diagonal incision is made about 4 inches in length, on a line connecting the anterior superior iliac spine with the umbilicus, the upper third to fourth of the incision being above this line. Another description of a very similar incision is: one extending from 1½ to 2 inches medial to the anterior superior spine and parallel to the inguinal ligament, in a person of average proportions, the upper limit of the incision being above the iliac spine. The fibers of the external oblique are divided in line with the skin incision, and the fibers of the internal oblique and transverse abdominis muscles are separated in their natural direction. The peritoneum is now opened, and the sigmoid is brought out of the wound. The sigmoid is then fed back into the abdomen until a taut point on the upper limb is reached. This takes up

the proximal slack, thereby permitting a more thorough resection during the subsequent operations and lessening possibility of prolapse later.

The colon is lifted up and held and avoiding its vessels a small incision is made in its mesentery or a hemostat is forced through it. This is best done between the bowel and the marginal artery as indicated in Figure 643. The peritoneum is grasped by the forceps, brought under the loop through the mesenteric orifice and united with 2 interrupted sutures. If the colostomy is to be permanent the fascia may be similarly joined; if the incision is large, the fascia beyond the loop may require 1 or 2 sutures. Then the skin is approximated as in Figure 643 *d* and *e*. There is much question as to whether the puckering suture, Figure 643 *a*, closing off the lateral peritoneal gutter by joining the lateral peritoneum to the mesentery, is required. Its purpose is to prevent a loop of small intestine slipping through and becoming obstructed. It is much less used than formerly.

As to better continence, there are no special colostomies which offer anything in this respect. None provide continence. Comfort depends upon the patient keeping himself somewhat constipated and using irrigations, sometimes daily, often every second day.

If the colostomy loop is not under much tension, gas and feces will be passed in a few days into and through the distal bowel and no puncture required. Puncture by scalpel or thermocautery may be done when required, preferably after forty-eight hours or more. To promote wound repair, delay cutting the loop until the sixth or seventh day if the patient's condition is favorable.

With cautery or scalpel cut the bowel transversely at the lower third of the loop to produce a small distal and larger proximal rosette.

Postoperative complications are hernia, prolapse of mucosa or bowel, stenosis of the stoma and retraction.

Mucosal prolapse is excised, several interrupted sutures attaching it to the bowel wall; the prolapsed bowel is amputated, the cut edges carefully approximated with interrupted sutures.

Stenosis and retraction are best met by dissection, freeing the bowel from the abdominal wall and excising a ring of skin ½ inch or more in width around it, then suturing the abdominal wall, and attaching the bowel, protruding about an inch, to the skin by interrupted sutures.

Ochsner, Jackson, Heyd are advocates of the Devine transverse colon colostomy for complete fecal diversion in certain cases of carcinoma of the left colon; Dixon and Ochsner favor it in anterior resection of the sigmoid. Devine's purpose is to "defunctionalize" the colon distal to the stoma.

Devine's method is to make a high right rectus incision midway between the costal margin and the umbilicus, bring up a loop of transverse colon and then incising the mesentery and the necessary vessels. The adjacent tænia coli are sutured over a 10 cm. distance and the bowel cut between clamps inserted through two 3 cm. skin incisions made about 3 cm. lateral and parallel to the original incision. One of the cut ends is brought out through the skin to the right and the other to the left of the primary incision, to provide complete fecal diversion. See Devine incisions illustrated in Figure 643. This has been modified by others in using a transverse incision. This incision is of real advantage in acute obstruction.

Wangensteen. has devised a loop colostomy to attain complete fecal diversion used in the right transverse colon and in acute obstruction as a temporary measure and in the sigmoid for irremovable cancer, ordinarily. A fairly generous loop of

bowel is brought out and the abdominal wall closed about it. Then instead of the one glass tube commonly used, two are placed under the loop, separated and held in opposite directions by rubber tubes attached to the two glass rod ends. The U shaped rubber tubes thus formed are held in traction by adhesive tape attached to the skin. An elliptical area of the bowel is excised and the two stomas thus made are held apart by the rubber tube-adhesive tape traction.

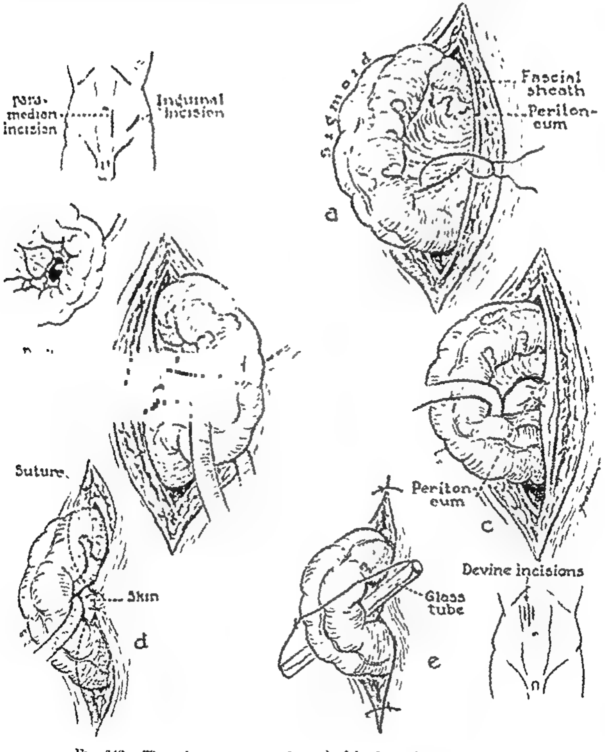


FIG. 643 —The colostomy suture shown in (a), above, is not imperative.

The foregoing applies particularly to loop colostomy. When performing a tube colostomy as in a Miles abdominoperineal resection, if the bowel is brought out about 3 inches and gauze wrapped, retraction is avoided; any excess of bowel remaining, can be excised by cautery in five to seven days without difficulty. The

less colostomies are complicated by excess suturing, the better; avoid sutures through the bowel wall.

Closure of Colostomy.—The proper time for restoring the continuity of the bowel lumen and closure of the colostomy depends upon the nature of the lesion for which colostomy was done; relatively soon, six weeks or so, in the case of an acutely obstructed carcinoma after resection of the neoplasm and healing has occurred; months afterward in the case of a slowly regressing inflammatory obstruction. When there is no spur and adequate intestinal wall, closure is made directly; if the spur exists, as it always does after exteriorizations, it must first be eliminated. Even when a spur is present mobilization of the bowel can be done and a secondary complete anastomosis of the bowel performed. This is rarely advisable, as it cannot usually be done extra-peritoneally. *As noted, exteriorizations are now infrequently* done.*

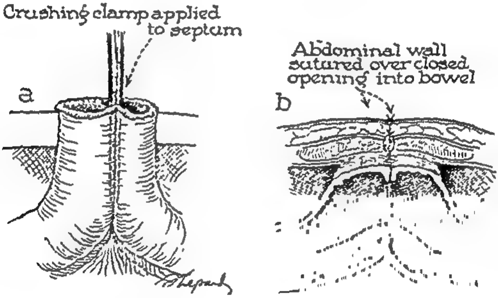


FIG. 644.—Closure of colostomy (*a*) crushing spur (*b*) closure.

Failure to Heal: This is generally due to incomplete destruction of the spur; obstruction in the bowel distal to the closure is an occasional cause, due, for example, to stricture, neoplasm, angulation from adhesions, at times fecal impaction.

After an obstructive resection or exteriorization operation many colostomies close spontaneously, however, union of mucosa to skin usually prevents closure.

Instead of the two or three weeks commonly awaited before application of spur-crushing clamps or forceps a delay of six to eight weeks or somewhat longer is advisable. This permits the inflammatory process and edema at the site to diminish and assures a better result.

A variety of spur-crushing clamps have been devised, Kelley Pean forceps are also effective, two being applied to destroy a V-shaped segment of the apposing bowel walls. This occurs in five to seven days, sometimes a day or two more. Tugging and manipulating the clamp to complete the process is avoided, as bleeding difficult to control may occur, or the bowel wall be torn.

Preoperative measures are carried out as for a resection and anastomosis: a low

* Indications vary but in general, the transverse colon is now used nearly as often as inguinal colostomy.

residue diet and irrigations through both loops for three or four days, and from noon of the preceding day opium in some form to quiet peristalsis.

Most colostomies can be closed extraperitoneally. An elliptical incision with pointed ends is made through the skin close to the bowel. The skin is dissected up,

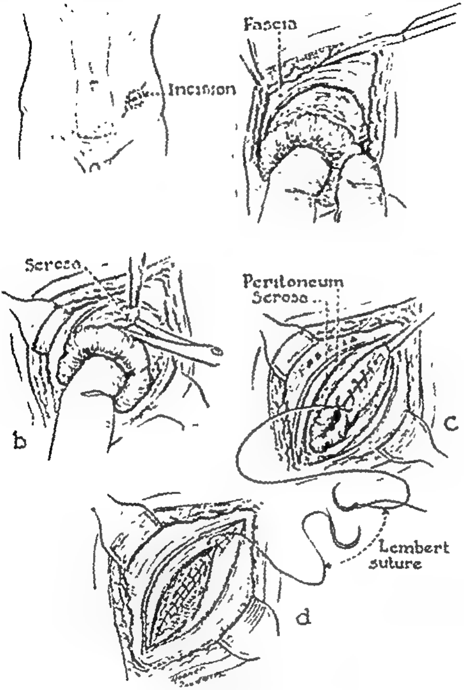


FIG. 645.—Colostomy closure; continuous suture is shown in c and d as it is advocated and often used. However interrupted sutures in both bowel and fascia are preferable, see text.

inverted over the bowel orifice and a silk or chromic continuous suture applied. Dissection is carried down to the fascia; it is cleanly but not widely exposed completely around the bowel, the peritoneum is then opened and the bowel lifted up into the wound. Manipulation is limited as much as possible in the peritoneal cavity; there are often adhesions sealing off the general peritoneal cavity fortunately. The bowel is closed transversely to its long axis by dissecting off the skin and creating clean edges for suturing.

Various methods of closures are used: (*a*) 1 row or interrupted mattress (Halstead); (*b*) one or two rows of interrupted Lembert sutures; (*c*) a mucosal closure by a continuous silk or chromic and an outer row of mattress sutures; or (*d*) a double row of continuous suture.

Two rows of interrupted silk or chromic catgut are used by the author and others.

The patency of the bowel is determined by grasping it with thumb and finger.

The fascia is closed with several interrupted chromic stitches, a small Penrose drain placed on the fascia and the skin margins loosely approximated. There is some serous drainage. Penrose drain is removed in forty-eight hours.

CECOSTOMY

It is rather generally accepted that cecostomy is indicated in (*a*) acute obstruction of the colon, (*b*) in chronic obstruction unrelieved by active medical measures and (*c*) in primary left colon resection, although there are dissentients who insist this is not necessary and that primary resection is safe without providing a safety vent,

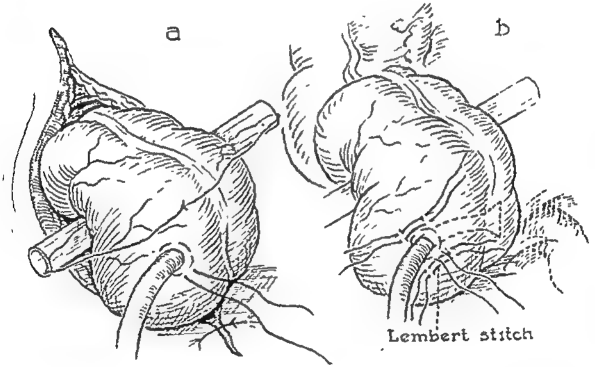


FIG 646.—Cecostomy

even when considerable feces are present, if distention is absent. A right-sided loop colostomy is preferred to cecostomy by some capable surgeons. Decompression is followed by resection in days or weeks, depending upon the degree of obstruction, or a cecostomy may be added to a primary resection at operation as a safety measure to avoid gas pressure at the suture line. In acute obstruction cecostomy or transverse colostomy should be done without exploration.

When used as a complementary measure in primary or obstructive resection the Witzel or Kader methods (Figs. 414 and 580) are satisfactory, as the intent is to prevent pressure from gas.

If fecal diversion is the requirement, exteriorization of the cecum is necessary. A size 28 or 32 catheter inserted, with purse-string suture, affords immediate gaseous relief and lessens the likelihood of wound infection. Spontaneous closure may be had in the Witzel and Kader cecostomies; this is especially true if the catheter is brought into the abdominal wall through a perforation made in the omentum. An operation for closure is often not necessary, even if the omentum is not used.

Because such cecostomies do not completely divert dejecta and thus fail to relieve obstruction, especially when the obstructive lesion is distal to the middle of the transverse colon, McNealy advocates mobilizing the cecum, if necessary, and bringing it out through the wound to provide effective diversion of the fecal content (Fig. 646). This is a satisfactory procedure; it usually requires a subsequent dissection of the cecum from the abdominal wall and closure.

The same method is followed as in the closure of a colostomy, except that the suture of the cecal orifice is simpler: (1) purse string suture; and (2) interrupted sutures burying this, only being required unless tube is brought out through omentum.

Transverse colostomy is now the general practice for all lesions of the right colon where colostomy is required. In acute obstruction the necessity of providing relief, "getting in and out" quickly, avoiding full exploration and undue manipulation are cardinal principles.

RESECTION OF THE CECUM AND RIGHT COLON

Ileocolostomy.—Nearly all carcinomas in the right half of the colon, *without symptoms of partial or advanced obstruction* can be treated effectively with relative safety by a primary resection. This presupposes proper preoperative management. Perhaps five per cent or less require two-stage procedures because of associated disease or advanced age. The same statement may be now made, in cancer of the transverse and splenic flexure of the colon.

A long right rectus incision having it's midpoint at or a little below the umbilicus is made to expose the field of operation.

Placing the patient in a moderate Trendelenburg position aids in exposure. Laporatomy sponges isolate the small intestine.

The object of this operation is to remove the cecum and ascending colon as high as the distribution of the middle colic artery and about 6 inches of the terminal ileum. The ileocecum is brought up and after ligating a few of the terminal branches of the mesenteric vessels just beyond the wall of the ileum, clamps are applied at an angle of about 45° to section the bowel obliquely, leaving its greater length at the mesenteric border. The latter to conserve blood supply and the oblique cut to give as large an ileal orifice in the stoma as is possible. Straight crushing clamps, Payr, Partipilo, Rankin or others may be used (Fig. 649). The bowel is divided between the clamps by cautery. The cecal end is closed by an inversion suture and dropped back. Crushing the bowel below the clamp, ligating it at the line of crushing, applying a purse-string suture and invaginating the end and tying the purse-string is a quick satisfactory maneuver.

On the anterior wall of the transverse colon at about the junction of its proxima and middle thirds, unless it is a case of carcinoma of the hepatic flexure, when a position farther to the left is chosen, a site for the proposed anastomosis is selected. An end-to-side is a satisfactory type at this site (Fig. 649); if lateral union is made,

care must be taken to avoid leaving a blind pocket in the bowel at the end of the stoma. If a lateral anastomosis is done: make the stoma about $2\frac{1}{2}$ inches long. Bring the bowel well up into the clamps so that as little blind pouch as possible will remain beyond the stoma. The ileum should point to the left. Complete peritonization cannot always be obtained; if it is not, a Penrose gutta percha drain may be left in for one or two days; its value is uncertain, it may eventuate in bacterial invasion along the drain from the abdominal wall.

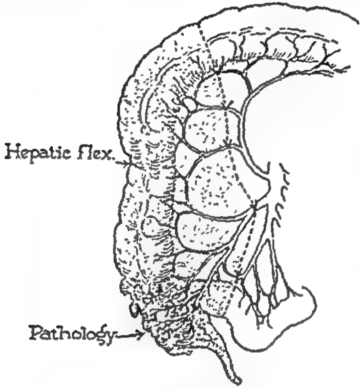


FIG. 647.—Schematic drawing showing the extent of excision and ligation of blood vessels for carcinoma of the cecum. Shaded area represents the amount of bowel and tissue to remove.

After completion of the foregoing, the abdomen can be closed and resection done later, or immediately as a one stage procedure if conditions permit. With a deep retractor on the right to expose the cecal region, and laparotomy sponges (packs) clearing the field of small intestines, the cecum is carefully drawn toward the midline by the assistant, while the cecum is mobilized. The parietal peritoneum is incised along the previously mentioned "white line" of union of visceral and parietal peritoneum if this is readily discernable, and if not, just lateral to the cecum and ascending colon, this region being quite avascular. The bowel is separated from the posterior abdominal structures by blunt dissection with the fingers or wiping with gauze, bringing involved lymphatics and some fat up with the posterior peritoneum toward the midline. The vas and spermatic vessels are exposed as dissection progresses, then the ureter is seen adjacent as it runs downward over the psoas muscle. Vas and spermatic vessels may be sacrificed if necessary. Excision of a portion of the ureter is sometimes necessary; usually it is not involved. Pinching the ureter with a hemostat just adjacent to its severed end and applying a firm silk ligature secures effective closure. Atrophy occurs in the occluded kidney; if infection is present in the organ at the time nephrectomy may be demanded later. The ileum and its lymphatic bearing tissue is readily brought up. As mobilization of the ascending colon proceeds, while the bowel is drawn toward the midline, the

retroperitoneal duodenum is exposed. Especial care is exerted here to avoid injuring it, since it is difficult to repair a duodenal fistula and, in these cases particularly, the mortality rate is high.

Dissection upward is completed and the gastrocolic omentum ligated and severed to a short distance beyond the anastomosis, and the omentum excised from this site downward.

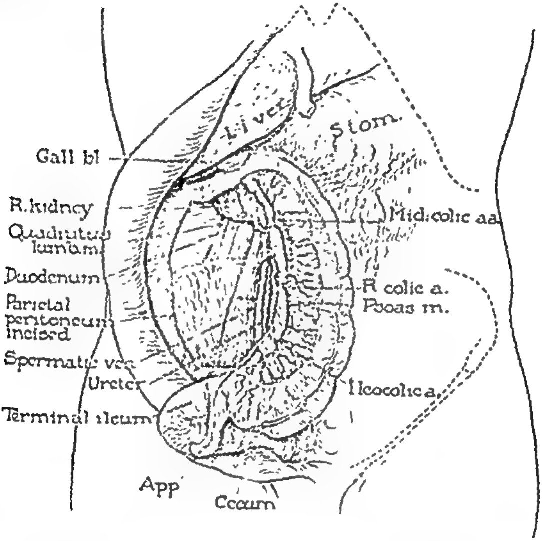


FIG. 618.—Illustrates the essential anatomy concerned in the resection of the cecum and ascending colon. Notice the relationship of the ureter and the spermatic vessels to the bowel.

When choosing the site for excision and subsequent anastomosis the relation of the middle colic artery to the transverse colon was ascertained, or as much information as possible was obtained by direct inspection, palpation or compressing it between the finger tips.

But only after the foregoing and on completing the mobilization is ligation of the main arteries done. The principal vessels are now identified. The mesenteric vessels may be clamped and ligated as encountered. However, the operation is shortened and simplified by clamping the principal vessels centrally, near their origin, toward the root of the surgically made "mesentery:" the right colic and ileocolic and the marginal arteries just proximal to the anastomosis. Close regard is given to the vessels near the anastomosis and ligation here is cautious, to assure an adequate blood supply.

Peritonization of the exposed retroperitoneal tissue is proceeded with, the posterior peritoneum being brought together by continuous suture (Fig. 649). Complete approximation cannot always be obtained. Serum accumulates in the retroperitoneal surfaces and drainage is usually desirable. A Penrose gutta percha drain alone may be left, but this drain with a thin wick of gauze and a split rubber

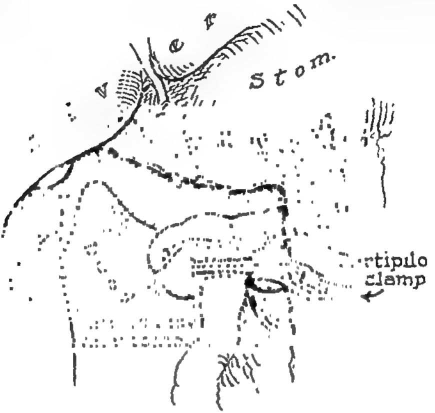


Fig. 649.—Ileocolostomy.

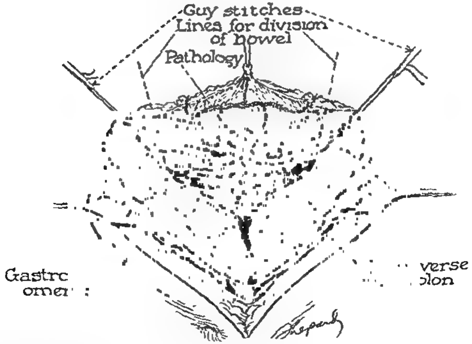


Fig. 650.—Mikulicz operation. The transverse colon (or sigmoid) has been freed from its attachments and blood supply to the portion to be resected has been ligated. Note that insufficient amount of mesentery is removed

tube is safer. The Penrose drain is removed in forty-eight hours and split rubber tube, forty-eight to seventy-two hours later, usually.

Mikulicz (Block, Paul, et al.) Operation.—The procedure generally designated as a Mikulicz, and which may be regarded as an obsolete operation, is a three-stage operation; consisting of (1) exteriorization of a loop of tumor-bearing colon, suturing the adjacent limbs of the loop thus made (2) excising the tumor a few days later and (3) after crushing the wall made by the approximated bowel—the spur, closing the colostomy some time later. It was a marked advance in its time, providing an extraperitoneal resection, lessening the danger of peritonitis. Its chief limitation was the high number of recurrences, some actually from wound implantation, others because carcinoma remained in the mesentery. When the mesentery of the loop is simply incised and its vessels ligated, the Mikulicz operation is not suitable for cancer cases, as involved mesentery usually remains.

It was applicable in resecting any part of the colon except the lower end of the sigmoid and is especially useful for resections of the sigmoid and transverse colon.

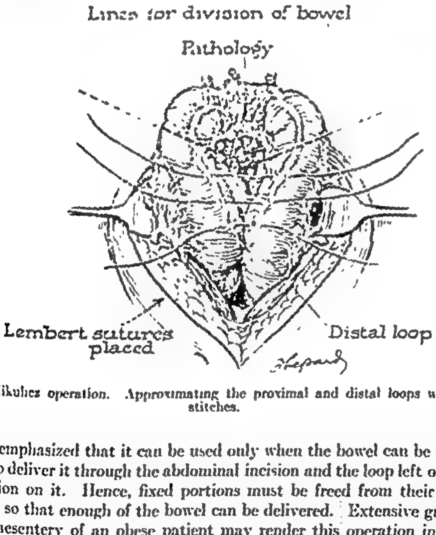


Fig. 651.—Mikulicz operation. Approximating the proximal and distal loops with Lembert stitches.

It must be emphasized that it can be used only when the bowel can be sufficiently mobilized to deliver it through the abdominal incision and the loop left out without undue tension on it. Hence, fixed portions must be freed from their peritoneal attachment so that enough of the bowel can be delivered. Extensive growths and the short mesentery of an obese patient may render this operation inapplicable.

Modifications of Mikulicz Operation.—Current surgical practice is concerned only with *modifications* of the Mikulicz operation, especially two: (1) Lahey's and (2) obstructive resection (Rankin); these are described although they are obsolescent, because one may rarely be indicated.

In both, a rather triangular portion of mesentery below the tumor is resected to remove the area of lymphatic spread. Both are accepted procedures in carcinoma

of the colon. Leahy used his method in all colonic cancer above the rectum. Most surgeons believe that resection and ileocolostomy is preferable in lesions of the right colon.

The *Lahey-Mikulicz* carries out the steps illustrated but the bowel ends are "staggered" when ileum and colon are united (Fig. 653). Three to 6 inches of ileum are sutured to a tænia coli and the end of the ileum projects well beyond the cut end *of the colon perhaps 3 or 3½ inches. This enables the introduction of a rubber or* glass tube in the ileal end. It is then tied in and a tube is led to a drainage bottle, thus permitting immediate release of intraintestinal pressure and serving to avoid wound contamination. In a right-sided carcinoma he sectioned the colophrenic (costocolic) ligament to bring the transverse colon to the right. In carcinoma of the

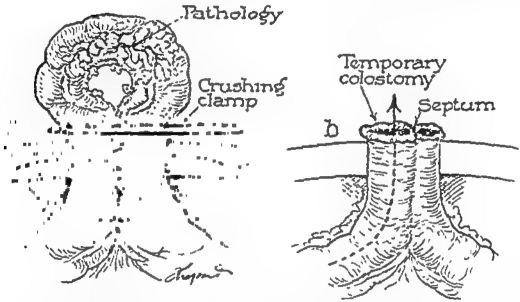


FIG. 652.—Second stage of Mikulicz operation. (*a*) Application of forceps at line of excision to excise the lesion by means of pressure necrosis (forty-eight to seventy-two hours later); (*b*) after the pathological segment sloughs a functioning colostomy remains.

splenic flexure he advised against a triangular resection of the posterior peritoneum, excised rather a segment of an ellipse, to avoid excision close to the jejunum and the ligament of Treitz, as the mesentery is thin here and tears on suturing.

In right colon cancer the Mikulicz procedure has not the widest acceptance, primary resection and ileocolostomy is more generally done.

Obstructive Resection.—This is a modification of the Mikulicz method. As Rankin was one of its outstanding advocates, his technique is given. It was his procedure in cancer of the left half of the colon if obstruction is absent or has been relieved. An integral part of it is an excision of as wide an area of gland bearing mesentery as is permissible. If obstruction is present a cecostomy or colostomy is first done; with the introduction of antibiotics he used the latter procedure more frequently.

Then at the subsequent operation the choice is between an obstructive resection or resection and anastomosis. He preferred the former usually, as it is safer.

First Stage.—An incision is made over the location of the neoplasm and exploration made. If it is in the transverse colon, the gastrocolic and greater omentum is dissected off; if in the sigmoid, as it usually is, the parietal attachments are incised and the tumor delivered Then the Trendelenburg position is obtained and the

intestines packed off by laparotomy packs. The loop of bowel is drawn well up, the vessels clearly seen, ligated so that a triangle, more or less, having its apex at the root of the mesentery, can be removed with the tumor. After making sure of an adequate blood supply to the remaining bowel ends, the Rankin or other intestinal

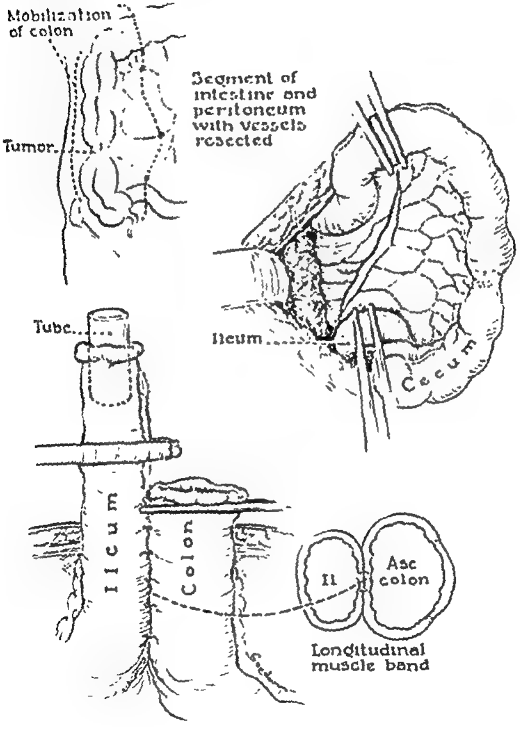


FIG. 653.—Lahey-Mikulicz modification.

crushing clamps are applied to the bowel. These are attached so the body of the instrument will lie on the abdomen, *e.g.*, from the right in sigmoid resection.

A rubber covered spring-clamp is placed above and the bowel between is cut by cautery. The divided mesentery is now brought together and closed by ties or suture, being careful not to injure vessels beyond the cut edge. The raw surfaces are peritonized, if possible. The bowel is brought out of the wound with its clamps, and

the wound is closed in layers, as usual. The peritoneum is now pulled through underneath the clamp and united with one mattress suture. No sutures are placed in the bowel. If mobilization has been limited the clamps are left down in the wound, and a gauze strip is packed closely about the bowel. After forty-eight to seventy-two hours of obstruction, the clamp on the proximal end of the bowel is opened or removed and that on the distal side left on until it drops off, which usually occurs about the sixth or seventh day. Avoid leaving clamps down in wound, if possible.

Second Stage.—Six weeks to two months is not too long to wait before application of spur crushing clamps. Various spur crushing clamps have been devised, several of which are satisfactory. Rankin uses two Kelley Pean clamps to take out a triangular portion of the spur with its apex downward. The septum is cut through in five to nine days.

Third Stage.—Closure is done as indicated for colostomy (Fig. 645). If done through a rectus incision repair of an associated hernia may later be necessary. This usually heals satisfactorily, in spite of the local contamination about the colostomy closure.

Largely because of antibiotic medication primary resection and anastomosis is now recommended on the right colon. Thus the use of modifications of the Mikulicz procedure has lessened.

CHAPTER 77

CANCER OF THE RECTUM

By Clement L. Martin

SPREAD OF CANCER

As the abdominoperineal resection of Miles has won the widest acceptance of all operations for cancer of the rectum and lower sigmoid, he is quoted at length. The low mortality and splendid end results achieved with it attest to its value.

In a classical paper (Surg., Gynec., & Obst., February, 1931) entitled "The Pathology of the Spread of Cancer of the Rectum and its Bearing upon the Surgery of the Cancerous Rectum," W. Ernest Miles made important observations as to the mode of spread of cancer of the rectum. He described the process of extension of the growth as follows:

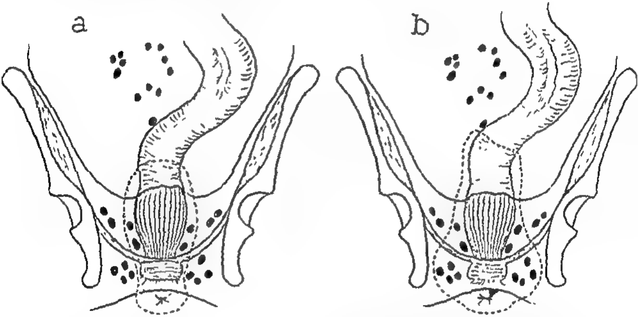
An adenocarcinomatous tumor of the rectum, when observed in an early stage, is confined to the mucous membrane and the submucous tissue. The tumor is sessile and is readily movable upon the subjacent coat of the bowel. It gradually increases in size, and during the process of growth, it may spread in three different directions: (1) by direct extension through the continuity of the tissues; (2) through the venous system; and (3) by means of the lymphatic system.

1. Spread, by direct extension through the continuity of tissues, takes place in two directions: on the mucous surface of the bowel, and through the thickness of the bowel. The marginal increase is generally greater and more rapid in the transverse direction than in the longitudinal axis of the bowel. It is not uncommon to find that, whereas nearly the whole of the circumference of the ampulla has been invaded, the extent of the growth longitudinally is less than 2 inches. The growing edge undermines the more normal mucous membrane extending in the submucous tissue deep to the muscularis mucosæ. Such surface extension is usually slow; probably taking six months for the growth to travel around a quarter of the circumference. Miles reported 4 cases which he observed in an early stage of development, and in which the patients either refused to submit to operative treatment, or were unable to submit to it for some reason or other. When first seen, the growths had not infiltrated the muscular coat, and were freely movable upon the subjacent muscular coat. At the expiration of one year the extent of the circumference of the bowel involved was five-sixths, four-fifths, and three-fourths, respectively. From this, Miles made the observation that by the time three-fourths of the circumference has been involved, the growth has existed for a period of eighteen months. He was of the opinion that it is after penetration of the fascia that the sacrum, uterus, or vagina, the prostate or bladder, can become invaded. Miles concluded that the spread of cancer is a comparatively slow process, and that direct invasion of the neighboring structures does not take place until at least one year after the appearance of objective symptoms, when at least three-fourths of the circumference of the bowel has become invaded.

2. Extension by means of the venous system occasionally occurs. According to

In a few cases, perhaps 5 per cent, when the neoplasm is at the rectosigmoid juncture or lower, even having its lower border at a point 10 cm. above the pectinate line of the anus, anterior resection can be performed. If it is nearer to the anus than 20 cm. it is more difficult and less rewarding, complications greater and recurrence more frequent. It should be done only when there is little or no gross inflammatory reaction about the neoplasm and no glandular involvement other than that removable with the excised tumor; this ordinarily means that the latter has not grown unduly large. Suturing deep in the pelvis is difficult; anastomosis below the rectosigmoid juncture is especially difficult.

Obesity is a contraindication generally; the procedure is less difficult in the type of pelvis found in a thin tall male.



...cted nature of Krake's operation The rectum is ... the upward and downward zones of spread. (*b*) ...he radical operation. The perianal skin and the ischiorectal fat are removed.

The thing which usually causes the death of the cancer patient one, two, three or *more years after operation is still cancer, so the elimination of the area of lymphatic* spread is still of primary importance. Desirable as it is to all of us, retention of sphincter function must be a secondary consideration.

ABDOMINOPERINEAL RESECTION

Miles' Operation.—This has been the procedure of choice for a number of years, in cancer of the rectum and lower sigmoid. Primary resection and anastomosis in carefully selected cases is somewhat lessening its indication; continuous spinal anesthesia is most satisfactory; inhalation anesthesia may be used. The operation is almost always done in one stage. Only serious functional impairment or shock not responding to treatment may favor a decision at operation to defer the posterior resection. The Trendelenburg position is used, *after a retention catheter* has been inserted in the bladder.

Abdominal Resection.—A left paramedian incision is made from the symphysis to the umbilicus or a little above it. A median incision is used by some surgeons.

Exploration is made with the left hand, from above downward, palpating the liver for metastases and the aorta, iliacs and region of the hemorrhoidal vessels for glandular involvement. If operable, the sigmoid is lifted up and the pelvis cleared by packing off the small intestine with two or three laparotomy sponges.

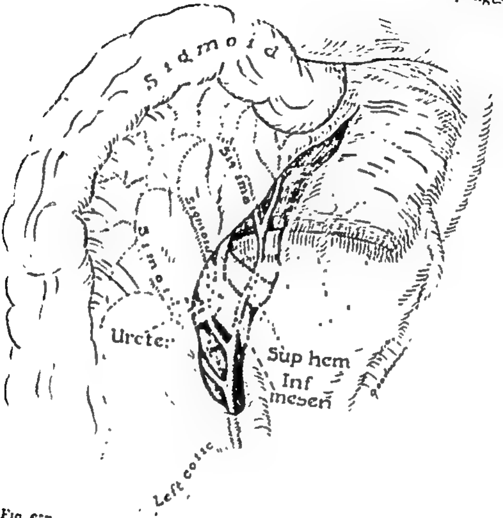


FIG. 657.—The relationship of the ureter to the left iliac artery.

The lateral peritoneum (mesentery of the sigmoid) is incised just beyond the bowel and extending down into the cul-de-sac, and partially encircling the rectum laterally and anteriorly. The left ureter is identified crossing the left iliac artery and running downward; it may be felt between the left thumb and finger or be seen to contract on stroking with a forceps. It often lies exposed in the peritoneal incision.

Adhesions generally present between the left side of the sigmoid mesentery and the iliac parietal peritoneum are incised to aid mobilizing the sigmoid.

(1) The superior hemorrhoidal vessels are now identified, ligated and cut, usually just below the bifurcation of the aorta. A helpful maneuver in identifying the vessels especially in a fat mesentery is to palpate them between left thumb and finger as the hand holds the bowel, the finger on the mesial, the thumb on the lateral surface of the mesentery. The ligation is usually distal to the point of origin of the sigmoid arteries, Fig. 657. The site of ligation varies depending upon the location of the cancer and extent of glandular involvement in the mesentery. Ligate as

high as is safely feasible, being careful to avoid the left colic artery, on which the blood supply to the bowel forming the colostomy depends.

The median peritoneal surface of the mesentery is now incised, along the bowel and joining its fellow from the opposite side of the anterior rectum.

(2) The rectum is now mobilized, this is easily done by passing the right hand into the hollow of the sacrum and bringing the bowel and its adjacent fat forward, staying close to the sacral surface and dissecting down to the coccyx. If bleeding

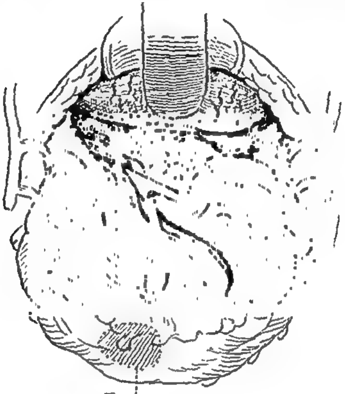


FIG. 658.—Incision of lateral peritoneum.

is free and this is rare, a hot laporotomy sponge may be left in a few minutes. This does not cause delay. As blunt hand and scissors dissection is carried forward it is limited by dense fibrous tissue laterally: the "lateral ligaments." These require incision, which is best deferred until the rectum is well separated anteriorly by finger dissection, pressure being made away from the rectum rather than toward it to avoid perforation.

(3) The "lateral ligaments" are now definitely identified (Fig. 659) and may be clamped and cut with long scissors after the clamps are removed or cut without clamping. The middle hemorrhoidal artery which is often in this fibrous tissue does not always require ligation. Rarely it does and the vessel is then clamped and ligated.

As the "lateral ligaments" are isolated by blunt finger dissection, aided by occasional use of long scissors, seminal vesicles can often be seen; these assist in orientation and dissection is carried down posterior to them. As dissection of the rectum from its adjacent tissue proceeds, the hand sweeps around its full circumference to make sure that it is adequately freed on all sides and deeply enough toward the anus. Then the loop of sigmoid which has been held taut by the assistant is inspected and after ascertaining bowel viability of the upper portion, clamps are

applied (Fig. 660). Wolfson clamps are preferable to DeMartel-Cope clamps, in my hands. Ordinary heavy Kocher or small Payr clamps can be used. The latter require a rubber hood over the cut end of the bowel, Fig. 661.

Two forceps are placed proximal to and a second pair is placed well below the growth so that only a small portion of the distal segment will remain after the bowel is excised. By removing a section of bowel there is less to place down in the infra-

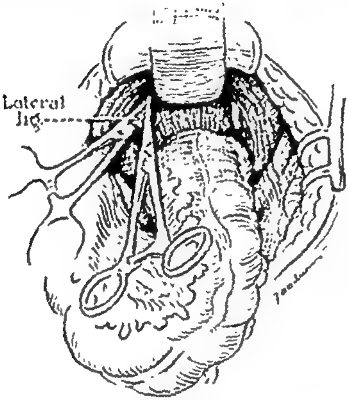


Fig. 659.—Ligation and excision of the lateral ligament

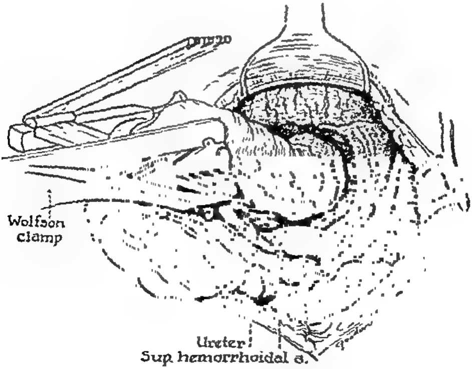


Fig. 660.—Wolfson clamp applied.

peritoneal space and closure of the peritoneum is facilitated. If the growth is very low in the bowel a segment may not have to be excised.

If Wolfson clamps are used the small clamp remaining on the bowel end provides its closure. *If ordinary heavy hemostats or similar clamps are used the bowel is* tied off by a loop of heavy silk and a thin rubber square about 4 x 4 inches tied over this as a cap.

The actual cautery is used to cut the bowel between the clamps; if the scalpel is used instead, phenol, then alcohol are commonly swabbed on the bowel edge in the clamps.

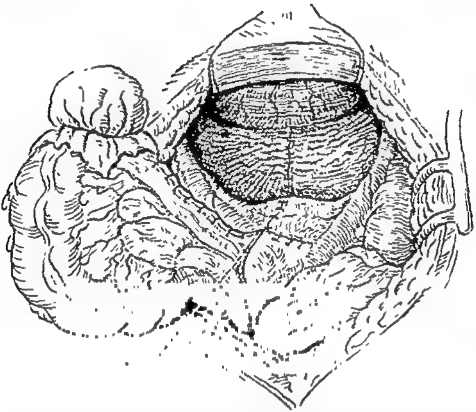


FIG. 661.—The abdominoperineal resection completed. The proximal segment is shown "capped" here to indicate an alternative procedure to the use of the Wolfson clamp

The bowel containing the cancer having been removed, the distal segment is folded down into the pelvis below the peritoneal plane and the peritoneum is closed.

If the peritoneum is scant and it is evident that it will not approximate readily, it can be freed by further dissection. Two or three hemostats are placed on its edge and as it is held taut by the assistant, dissection is made underneath by the fingers; a few snips of the scissors may be necessary where fibrous portions of the infraperitoneal fascia require.

Posterior Resection.—*Time should not be lost here; the blood pressure often* drops at this phase especially if the preceding work has been prolonged; intravenous fluids, usually started when the operation was begun and circulatory stimulant medication must be used with understanding and discretion. If continuous spinal anesthesia is used the needle can usually be withdrawn and the operation completed without further anesthesia.

As stated previously, the posterior resection may be done immediately or later. If a one stage operation, proceed as follows:

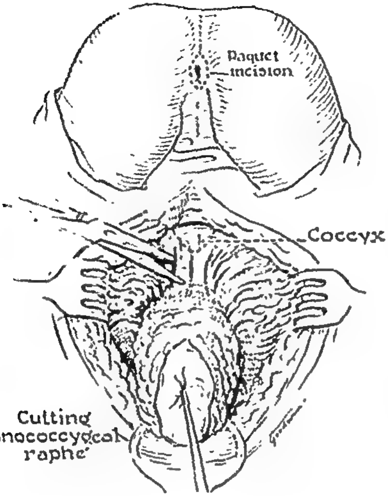


FIG. 662.—Second stage of abdominoperineal resection. Excision of anococcygeal raphe.

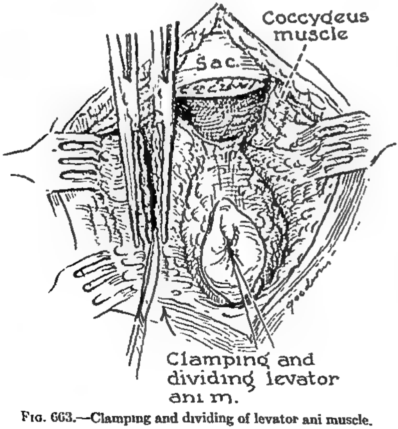


FIG. 663.—Clamping and dividing of levator ani muscle.

Place the patient in the prone position and then elevate the hips and tilt the table to secure the reverse Trendelenburg (Depage) posture i.e. head lowered, hips elevated, thighs separated, thighs and legs lowered.

(1) Close the anus with a suture of fish-line or heavy silk. If it can be done expeditiously it is well to swab the rectum out with moist gauze beforehand and then swab it with merthiolate or other antiseptic solution. A raquet incision is made, the circular portion around the anus, the handle in the midline extending to the level of the second sacral segment or thereabout. Undercut the skin and subcutaneous fat, controlling bleeding as encountered to open the operative field, from the upper to lower margin of the wound. Deepen the incision to include all of the ischiorectal fat.

Place a Beck goiter retractor, spring retractors or have assistants retract skin to provide exposure.

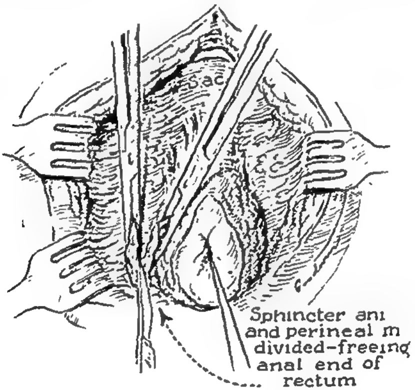


Fig. 664.—Clamping and division of sphincter ani and perineal muscles.

(2) Mobilize coccyx; this may be done by incisions between sacrum and coccyx to open the joint and an incision on the anterior surface joining them, then bending or fracturing and bending the coccyx posteriorly, or the dissection may be carried further and the coccyx excised. It is rarely found necessary to excise it. In the occasional instance, if it is left in, it may have to be removed later because of necrosis from deficient blood supply. Mobilizing the coccyx and cutting the coccygeus muscles aids in opening the operative field.

(3) An inch or inch and a half transverse incision is made into the dense connective tissue just below the sacrum, the fingers are then inserted to detach from the ventral sacral surface the fibrous and fatty tissue. This incision opens the fascia propria of the rectum and the bowel proper is thus exposed. By blunt finger and hand dissection the line of cleavage thus obtained is carried upward and the infrapelvic distal segment of the resected bowel is grasped, brought downward and delivered into the wound.

(4) Holding the bowel taut with the left hand, traction puts the levatores ani under tension; they are cut well out toward their origin from the wall of the pelvis, away from the rectum. If these muscles are not readily felt and exposed it is because the lateral ligaments were not sufficiently cut from above; then these may

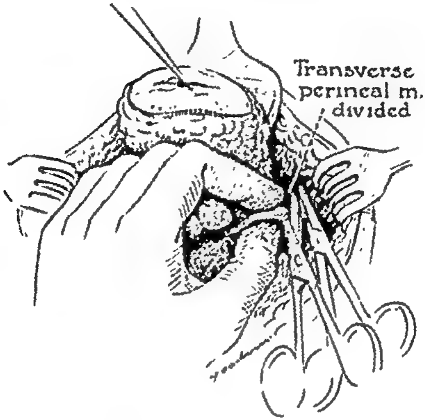


Fig. 665.—Division of transverse perineal muscle.

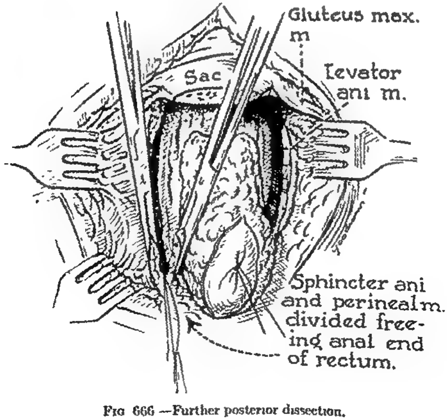


Fig. 666.—Further posterior dissection.

Place the patient in the prone position and then elevate the hips and tilt the table to secure the reverse Trendelenburg (Depage) posture i.e. head lowered, hips elevated, thighs separated, thighs and legs lowered.

(1) Close the anus with a suture of fish-line or heavy silk. If it can be done expeditiously it is well to swab the rectum out with moist gauze beforehand and then swab it with merthiolate or other antiseptic solution. A raquet incision is made, the circular portion around the anus, the handle in the midline extending to the level of the second sacral segment or thereabout. Undercut the skin and subcutaneous fat, controlling bleeding as encountered to open the operative field, from the upper to lower margin of the wound. Deepen the incision to include all of the ischiorectal fat.

Place a Beck goiter retractor, spring retractors or have assistants retract skin to provide exposure.

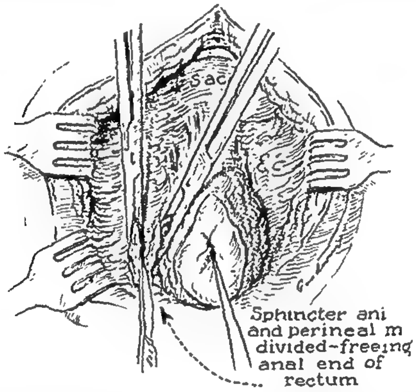


FIG. 664.—Clamping and division of sphincter ani and perineal muscles.

(2) Mobilize coccyx; this may be done by incisions between sacrum and coccyx to open the joint and an incision on the anterior surface joining them, then bending or fracturing and bending the coccyx posteriorly, or the dissection may be carried further and the coccyx excised. It is rarely found necessary to excise it. In the occasional instance, if it is left in, it may have to be removed later because of necrosis from deficient blood supply. Mobilizing the coccyx and cutting the coccygeus muscles aids in opening the operative field.

(3) An inch or inch and a half transverse incision is made into the dense connective tissue just below the sacrum, the fingers are then inserted to detach from the ventral sacral surface the fibrous and fatty tissue. This incision opens the fascia propria of the rectum and the bowel proper is thus exposed. By blunt finger and hand dissection the line of cleavage thus obtained is carried upward and the infrapelvic distal segment of the resected bowel is grasped, brought downward and delivered into the wound.

require further division. The latter are deep in the wound but hemorrhage from this source is rare, if it occurs the vessel is clamped and ligated.

(5) Now dissection is from below upward, carrying an incision outside the external sphincter ani, compressing the superficial and deep transverse perineal muscles between thumb and finger as indicated in the drawing, clamping and then *dividing them.*

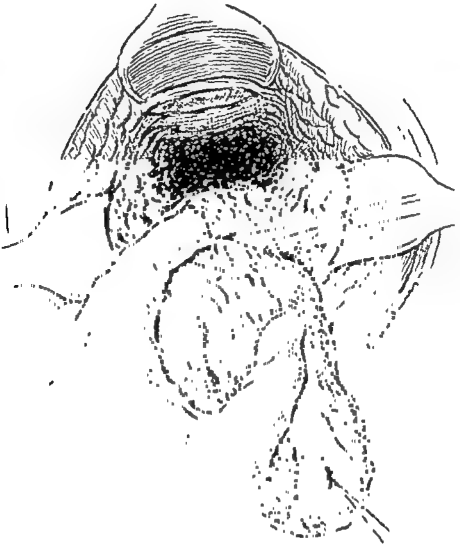


Fig. 667.—Dissection of rectum from anterior structures.

(6) Palpate the catheter in the membraneous urethra (Fig. 668) and by scalpel or scissor dissection separate the rectum from the prostate. This is a difficult part of the operation, the most difficult, in my opinion, if the patient is a male. Care must be taken to avoid incision into the rectum, as dissection is carried upward to and above Denonvilliers fascia between the rectum and prostate. After this is passed a line of cleavage is reached where blunt dissection is easily accomplished.

(7) As blunt dissection has been made from below upward it is a simple matter to hold up the rectum and separate it from its remaining fibrous attachments.

An 8 x 10 sheet of thin rubber (gutta-percha) is packed in surrounding a heavy laparotomy sponge or vaginal pack, and the wound closed after inspection to make sure of hemostasis, by two or three interrupted silkworm-gut sutures and other silk

skin-approximation stitches. Several inches of the pack is removed on the second post-operative day and it is out completely on the third post-operative day. The retention catheter in the bladder is ordinarily removed on the third post-operative day.

The proximal segment is either sutured into the original abdominal incision to provide a left rectus colostomy or brought out through a stab wound laterally to make an inguinal colostomy. The former saves time and is generally satisfactory, the latter lessens the danger of laparotomy wound contamination. About an inch and a half of bowel end is allowed to protrude above the abdominal wall. A dry gauze sponge wrapped around it holds it in place, no sutures are placed in the bowel. If the Wolfson clamp is used it is removed on the second or third post-operative day. The abdominal wall closure is made in the usual manner.

PRIMARY ANASTOMOSIS IN THE CANCER OF THE UPPER RECTUM AND RECTOSIGMOID

Although the Miles abdominoperineal resection has by far the widest application of all operations for cancer of the sigmoid and rectum, primary resection and anastomosis can at times be done with a good result. In well selected cases Dixon

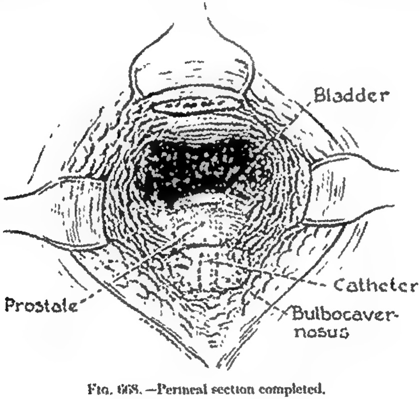


FIG. 668.—Perineal section completed.

has over a twenty-three-year period obtained a satisfactory number of cases with freedom from recurrence for three to five years in more than 200 operations. His procedure, anterior resection, is described.

The abdomen is entered through a lower right rectus incision, Trendelenburg position secured and after exploration laparatomy sponges placed, and the sigmoid loop delivered, its peritoneum is incised laterally and medially and the rectum separated from the sacrum by hand dissection (see Figure 669). It is often necessary to incise the lateral peritoneum of the colon as high as the splenic flexure to aid

and cutting and amputation of a small portion of the proximal segment to permit better approximation of the bowel ends. An end to end anastomosis is made with one or preferably with two rows of interrupted sutures and the peritoneum brought together above the intestinal suture line with a continuous suture (Figure 672), leaving it situated higher in the pelvis. A Penrose drain is inserted in the hollow of the sacrum and brought out through the abdominal incision.

After completion of the pelvic surgery a transverse colostomy is performed; through a small (stab) wound a knuckle of transverse colon is brought out and a few interrupted sutures placed as required in the abdominal wall. Petrolatum gauze is used to seal the colostomy wound around the bowel. It is punctured by cautery in twenty-four hours and closed three to six weeks later.

This operation has progressively earned a wider application. The antibiotics have had no small part in this.

It should not be done when the lower border of the carcinoma is distal to a point 17 cm. above the anus, except in the occasional case. And it should not be done

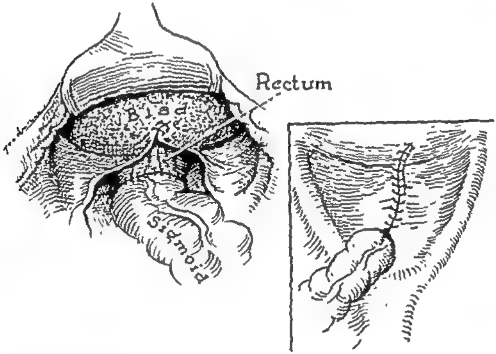


FIG. 672.—Anterior resection. The proximal and distal ends anastomosed and peritoneum closed.

when there is more than local extension of the neoplasm. Where the growth extends below the peritoneum it is not indicated, in my opinion.

In an effort to avoid the objectionable colostomy its application is often overstretched and early recurrence is not uncommon, either at the line of anastomosis or in nearby tissue.

ABDOMINOPERINEAL PROCTOSIGMOIDECTOMY WITHOUT COLOSTOMY AND WITH PRESERVATION OF THE SPHINCTER MUSCLES
(Babcock-Bacon's Operation.)

Technique.—*Abdominal phaze:* The abdomen is opened through a left oblique incision 3 centimeters above the inguinal ligament, beginning at a point to the right

of the midline above the pubic spine and ending medial to the left anterior superior iliac spine. Ordinarily, the left anterior rectus sheath is divided, and, in a few instances we have detached both the rectus and pyramidalis muscles from the pubic spine as suggested by Cherney. More recently we have returned to the classical left paramedian incision. The liver is examined for metastasis; the median lumbar, upper and lower mesocolic areas palpated for nodules, and the extent of the growth

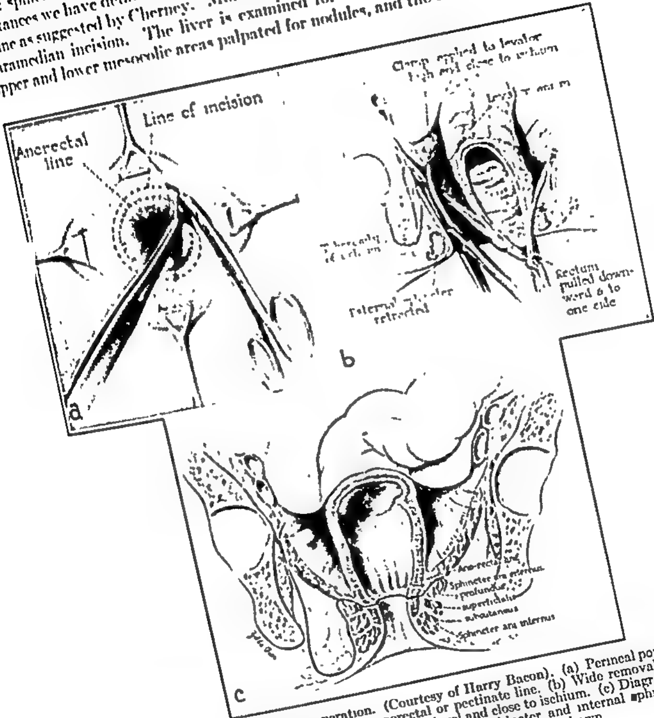


Fig 673.—Babcock-Bacon operation. (Courtesy of Harry Bacon). (a) Perineal portion of the operation. Incision carried ½ inch below anorectal or pectinate line. (b) Wide removal of levator muscles can be achieved by placing clamp at high level and close to ischium. (c) Diagram showing preservation of anal epithelium, 3 bundles of external sphincter and internal sphincter. Sacrifice of bowel with ischiorectal fat, levator and pelvirectal tissue is shown

determined. The patient is placed in the exaggerated Trendelenburg position and the pelvic cavity is cleared of small intestine by hot packs. The left lateral leaf of the mesosigmoid is freely divided wide of any malignant infiltration, the incision being carried downward to the rectovesical or rectouterine sulcus. In the course of the dissection, the left ureter, iliac, and the spermatic or ovarian vessels are exposed.

The spermatic or ovarian vessels may be divided and ligated. The sigmoid with attached fat and mesosigmoid is mobilized toward the midline thus clearing all gland bearing areas in the vicinity of the iliac vessels. The peritoneum on the mesial side of the sigmoid is incised downward, continued around the right pelvic brim, and across the sulcus between the rectum and bladder or uterus to meet its fellow of the opposite side. By gently inserting the hand into the postrectal cellular space in the pelvis, the lower pelvic sigmoid and rectum can be stripped from the anterior surface of the sacrum as far as the sacrococcygeal articulation. The lateral ligaments are rendered prominent and divided. They may or may not require ligation. Anteriorly, the rectum is separated in the female from the upper and mid portion of the vagina, and in the male from the base of the bladder as far as the prostate. Care should be exercised to avoid injury to the seminal vesicles and vas deferens. Ordinarily, transillumination is employed to visualize the inferior mesenteric, superior hemorrhoidal, and sigmoidal vessels, and their communicating arcades. By such, greater precision can be exercised of those to be preserved, which is essential for that portion of the sigmoid to reach through the perineum. The necessary vessels are clamped, divided, and doubly ligated. The arterial supply has been discussed in detail under anatomy.

The ligature is usually placed between the first and second sigmoidal branches. The point of viability is determined by observing pulsating arteries or by the character of the bleeding when the small vessels on the surface of the bowel at the level of resection are incised. Two and one-half grains of sulfathiazole powder are dusted over the viscera which is covered by the great omentum. More recently we have employed 100,000 units penicillin instead. The peritoneum may be closed with a continuous suture of No. 0 chromic catgut, fascia with interrupted sutures of No. 32 gauge alloy steel wire and skin with No. 35 wire, although our preference is figure-of-eight wire suture after the method of Smead.

Perineal Phase: Step 1.—The patient is changed to the lithotomy position on the specially designed spinal mattress, and the rectum packed loosely with antisepticized gauze.

Step 2.—The anal margins are clamped precisely in four divergent quadrants with Pennington hemostats and retracted.

Step 3.—A circular incision is carried just through the anal skin (squamous epithelium) one-eighth inch distal to and below the anorectal (pectinate, dentate) line.

Step 4.—The edges of the proximal incised skin are held taut from which is teased the surrounding musculature by sharp and blunt dissection. This muscle—the subcutaneous bundle of the external sphincter is readily prominent while medially the lower margin of the internal sphincter is evident. Briefly stated, retraction of the anal margin has partially displaced and stretched this musculature but the anatomic relationship remains unchanged. A review of the sagittal section on page 37 figure 33 (Bacon, 2nd edition) will disclose the fact that the internal sphincter muscle descends for approximately two-thirds the length of the anal canal—and is separated from the external sphincter below (subcutaneous bundle) by the intersphincteric line, perhaps better termed the "intermuscular septum." The initial incision which is made immediately below the anorectal line becomes displaced by virtue of the retraction. It is relatively easy to separate and introduce a small retractor into this septum between the internal sphincter medially and the subcutaneous bundle situated below and laterally. This must not be done because the internal sphincter would be sacrificed. Instead, both the subcutaneous bundle

of the external sphincter and the internal sphincter are dissected free and retracted gently about the entire circumference.

Step 5.—The four Pennington clamps used to retract the anal margin are discarded and fresh ones applied coronally to the gut edges of the bowel in a fashion as to occlude the lumen. These clamps are held together with a stout rubber band and drawn taut.

Step 6.—The dissection is carried cephalad between the thin rectal wall medially and the internal sphincter laterally until the increment or body of this muscle becomes thinned; in other words where it continues above as the circular coat of the rectum.

Step 7.—At this point the circular muscle is pierced and the longitudinal muscle divided together with a portion of the fibromuscular ring in order to enter an ill-defined cleavage plane between the levator muscle situated medially and above, and the deeper portions of the external sphincter (superficial bundle and profunda bundle) below. Thus, are the Pennington hemostats clamped to the rectal wall with attached levators for sacrifice, while the three bundles of the external sphincter and the internal sphincter have been separated for preservation.

Step 8.—Richardson retractors holding the musculature to be preserved are sharply angulated into the ischiorectal fossa to permit wide removal of fat therein.

Step 9.—Posteriorly, a single transverse incision through the fascia propria, which is closely adherent to the periosteum of the lower border of the sacrum is all that is needed to mobilize the rectum.

Step 10.—Anteriorly, the superficial and deep transverse perineal muscles are retracted and the line of cleavage cautiously followed between the rectum and prostate until the depth of the abdominal dissection is encountered. The seminal vesicles and vas deferens are again visualized. In the female, the rectovaginal septum is separated by blunt and gauze dissection until the divided portion above is reached. Mobilization has now been effected posteriorly and anteriorly.

Step 11.—By making traction on the bowel, the levator muscles are placed on the stretch, provided of course the lateral ligaments were divided during the abdominal phase. The levators from front to back are clamped high and wide with large curved hemostats; they are then divided, and ligated.

Step 12.—Mobilization being complete, the rectum and lower sigmoid are drawn through the wound and enclosed in a sterile towel. The segment of viable bowel must protrude well beyond the anal margin.

Step 13.—The pelvis is inspected for bleeding points and an antero-lateral pelvic floor is established by introducing two or three catgut sutures through the edges of the peritoneum.

Occasionally this closure is made from the abdominal phase although in our experience with 373 cases, the approximation effected is more precise from below. At no time is it advisable to place sutures between the peritoneum and the bowel although it is permissible to tack the fat tabs to the edges of the peritoneum.

Step 14.—One or two alloy steel wire sutures are placed interruptedly to approximate the stumps of the anterior levators. The sphincter muscles, which have not been incised nor divided in any phase, are permitted to assume their normal position.

Step 15.—A curved, perforated, metal drain is inserted posteriorly along the sacrum to evacuate blood and serum during the first twenty-four hours. A slit-dressing sutured with tincture benzoin is applied; the extruding bowel slit at a point 7 centimeters from the anal margin to allow for retraction and the bleeding

points ligated at this level. A mushroom catheter is inserted into the lumen of the bowel to a point above the new pelvic diaphragm and held in place by the Daniels clamp. The metal drain is removed at the expiration of twenty-four hours. Irrigation of the bowel through the catheter is begun the morning after operation using a few ounces of warm saline solution, at four hour intervals. It is continued until the time of the first satisfactory evacuation which is usually the third day. The clamp is then removed and the catheter withdrawn.

Bowel Stump.—*Management of redundant or protruding bowel stump.*—The portion of the bowel distal to the Daniels clamp will slough-off because of pressure necrosis. The remaining gut will ordinarily retract to the anal margin and "draw-up" the anal epithelium to form a normal anal canal and normal anorectal line. If such does not occur the patient is taken to the operating room on the seventh or eighth day and the bowel removed.

Technic.—Using the jack-knife position with the patient under sodium pentothal or spinal anesthesia, the anal skin, which has become puckered and rolled, is teased from the bowel, gently mobilized and retracted. The gut is drawn taut and a Payr clamp placed coronally about the bowel which is excised with the Paquelin cautery. The edges of the freed anal skin are then lifted to their normal location and tacked to the edges of the mucous membrane of the bowel using fine catgut in mattress fashion. The foregoing is Bacon's description of his operation.

The Hochnegg "pull through" operation and Babcock's and Bacon's modifications of it have not won general acceptance. In the hands of the two latter surgeons the reported results have been good. It is, however, a less radical removal of the lymphatic drainage area than a Miles abdominoperineal resection. It avoids the abdominal colostomy and is thus more acceptable to some patients, but most patients are compelled to wear a perineal dressing after operation to absorb seepage and must use an enema every day or two to regulate fecal discharge. I have yet to see one who does not. "It does not preserve sensation and anal continence for liquids" (*Lahey*). *Minimal recurrence incidence and not the patient's preference* should ordinarily be the deciding factor.

POST-OPERATIVE COMPLICATIONS AFTER RESECTIONS OF THE RECTUM AND SIGMOID

The most common of these is atony of the bladder, evidenced by retention.

It is inevitable that some damage to the nerve supply occur; the presacral sympathetic plexus lying on the pelvic floor is often divided and frequently some parasympathetic fibers from the lateral pelvic wall. Prostatic hypertrophy may be present. Perhaps the majority of these patients have some urinary complication so close urologic coöperation is advisable.

Bladder drainage by a closed irrigation system is used. Early removal of the retention catheter, preferably on the third day, the free ingestion of water and encouraging the patient to void voluntarily lessen bladder dysfunction and is necessary. The bladder should not be distended beyond 500 cc., reinsertion of the retention catheter may be necessary. On removal, interval catherization and a daily irrigation is advisable. Intestinal obstruction may occur from various causes, among others: a fibrinous exudate kinking a loop of intestine; ileus of a portion of the small bowel, generally, I think, from operative trauma; or perhaps from a loop

of intestine slipping down the gutter lateral to an inguinal colostomy; the last is quite rare.

It may develop early in these cases, before the colostomy starts to function. If nasogastric tube drainage does not relieve in twenty-four or forty-eight hours a Witzel or other enterostomy should be done through a right paramedian incision in the first loop of distended small bowel presenting. This generally provides relief and the colostomy functions in three to five days; a week or ten days later after the colostomy is properly functioning the catheter in the bowel is withdrawn resulting ordinarily in a spontaneous closure of the opening. Early closure is favored by passing the enterostomy catheter or rubber tube through a small opening made in the omentum thus interposing the latter between the bowel and the abdominal wall.

Stenosis of the Colostomy Stoma.—In the occasional instance contraction of the skin and fascia produces a stenosis of the opening; it may be impossible to insert the tip of the little finger and irrigations, especially their evacuation becomes more difficult. It is generally caused by cutting the segment of colon too short, or in a double-barrel variety by making the proximal limb too short. It is favored too by a thick fat abdominal wall or pendulous abdomen. It can be repaired by exicising an inch or more of the cicatrix above (cephalad) the stoma. After incising the skin, the scar tissue below is divided carefully and deeply enough so that the finger enters the bowel and feels, after dilatation, a free passage through the abdominal wall. The peritoneum need not be opened ordinarily, but if it is some adhesions are frequently found around the colostomized bowel, partially or completely isolating it. Two or three interrupted sutures attach the freshened mucosal edges to the skin edge. The skin above is approximated with a few interrupted sutures, care being taken to allow adequate space near the bowel. The patient may leave the hospital in a day or two. This gives a permanently satisfactory result generally, but I have operated twice on some cases, three times on another, using various procedures. As long as there are satisfactory evacuations, no surgery is required, even if the little finger will not enter the stoma very little mucosa is seen.

CHAPTER 78

ANORECTAL DISEASES

By Clement L. Martin

ANATOMY

The most important anatomical structures of the anus and rectum are illustrated in Figure 674.

The *pectinate (dentate) line* is formed when the anal plate and cloacal membranes fuse and disintegrate, leaving a serrated margin. This is a true mucocutaneous junction and a landmark of surgical importance. The *intersphincteric (Hilton's) line* is unimportant.

The *anal crypts* are at the upper margin of the pectinate line; they lie between the columns of Morgagni and their inferior surface is formed by a fold of mucosa somewhat like a semilunar valve. Small ducts (*anal ducts*) extend out of some of the crypts, into the submucosa and muscularis; they are tubular or branching and lined by stratified squamous epithelium and in their depths by one or two layers of columnar epithelium. They are of consequence as a source and route of infection.

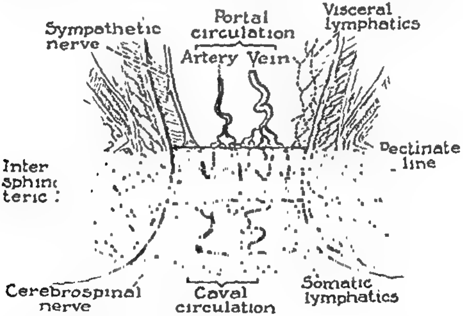


Fig. 674.—Surgical anatomy of the a n.

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sympathetic and parasympathetic systems through the second, third and fourth sacral nerves.

The *external sphincter* is larger, more readily palpated and just distal to the internal sphincter. It is a voluntary muscle but with some involuntary action. Ordinarily in a state of moderate tonic contraction to keep the anus closed, this tonus can be increased or decreased by voluntary control, or involuntary response to an unexpected external stimulus. It is chiefly supplied by the inferior hemorrhoidal branch of the internal pudendal nerve and the fourth sacral nerve. The structures immediately about the anal canal are innervated by these two nerves.

The levator ani and external sphincter, striated muscles, are supplied by somatic nerves; the internal sphincter and the rectum, smooth muscle, by visceral motor fibers. The mucosa of the rectum is believed to be supplied by visceral sensory fibers. Thus, rectal polyps can be fulgurated and internal hemorrhoids injected without pain; *inflammation, abscess, etc., in and about the anus are painful.*

ANORECTAL DISEASES

The four most frequent ano rectal diseases are considered. They are described in a much more detailed and discursive manner than lesions in the preceding section on the large bowel. This is done as the technic is simple, except in the more complicated fistulas, but effective results are dependent upon a thorough understanding of each particular disease and the exact diagnosis.

ANORECTAL ABSCESSES

Ano rectal abscesses are common and are the result of inflammation in the anus, rectum and adjacent tissues and organs, as well as from trauma either internal, *e.g.*, perforation by fish bone, instrumentation, abuse by sexual deviates, etc. or external from falls, blows or kicks. The symptoms are those of an abscess elsewhere, that is, fever, leucocytosis, pain, tenderness and other localizing evidences depending upon the involved site. Treatment consists of incision as soon as the diagnosis is made. In all cases, except the cutaneous, the patient is informed that a fistula may result, if not already present. These are classified, on the basis of location, as follows:

Infralevator (a) Cutaneous (integumentary)
(b) Subcutaneous
(c) Ischiorectal
Supralevator (a) Retrorectal
(b) Pelvirectal
(c) Submucous (mural, interstitial)

Infralevator Abscess.—Cutaneous or integumentary abscess is initially nothing more than a pustule. The subcutaneous (marginal, perianal) abscess is ordinarily a tender swelling at the anal margin and usually posterior or lateral to it. It generally arises from an infected crypt or fissure. An abscess just posterior to the anus is often termed post-anal, one anterior to it, a perineal abscess. The treatment of this type of abscess is by a vertical linear incision.

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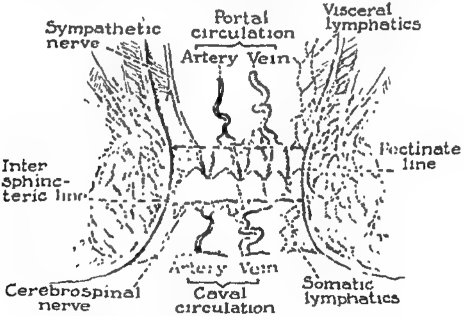

Fig. 674.—Surgical anatomy of the anorectal region.

The *anal canal* extends from the pectinate line outward to a somewhat indefinite boundary, the anus, anal verge or rima, that is, where the skin at the external margin of the canal naturally approximates. The canal is 1.5 to 3 cm. long and is lined by skin. *Anal papillæ* are knob-like or triangular projections of modified skin at the pectinate line, 3 to 6 in number usually and 3 or 4 mm. in length.

The *internal sphincter* is the thickened expanded end of the circular coat of the bowel, an involuntary muscle supplied through fibers of the intrinsic plexuses of the

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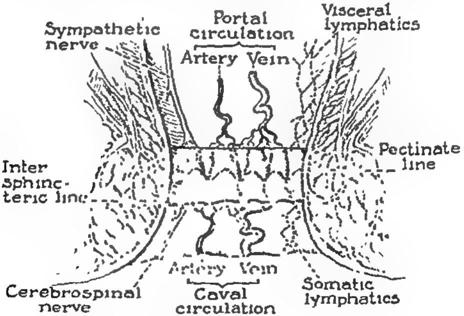

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Supralevator Abscesses.—Submucous abscess, as the name implies, is in the submucous layer of the rectum. It usually results from a cryptitis in the anterior or anterolateral anal wall and may be palpated as a soft swelling. It is not frequent. It may be localized or it may communicate with the pelvirectal, retrorectal or ischeorectal spaces. It is treated by a vertical fulguration incision of its lower half. This is preferable to incision as it lessens the danger of bleeding. This provides adequate drainage for an uncomplicated submucous abscess and the wound, will heal. If extension to the peri-rectal spaces, *e.g.* pelvirectal, etc., is present drainage of these is required. The treatment of abscesses in these spaces follows.

Superior Pelvirectal Abscess.—This is an abscess in one of the two pelvirectal spaces. More often the site of the primary infection is the cervix and adnexia, prostate, seminal vesicles or urethra; however, rectal disease is not an unusual cause, *e.g.* trauma, stricture, rectal inflammation and faulty hemorrhoid injection therapy.

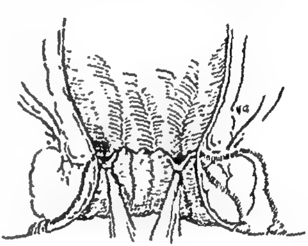

FIG. 677.—Cryptitis. Shows common route of infection from a cryptitis. The subcutaneous close to the sphincter fibers close to the skin (on the left) is the least common.

Symptoms.—The symptoms are those of a patient having an abscess, or at least an inflammatory process, evidenced by malaise, anorexia, fever, leucocytosis and perhaps chills. There may be no rectal complaint. As the abscess progresses, rectal fullness or a sense of heaviness may occur, while defecation is generally not painful. Genitourinary symptoms are not infrequent, *e.g.* an aching sensation in the groin or suprapubic region, frequent urination. Perianal symptoms are absent. The lower abdomen is tender to pressure, tense, or even rigid, on the involved side if the abscess is unilateral. These cases can be confusing especially in males. An acute appendicitis may be suggested. Abdominal findings in a man simulating a broad ligament infection in a female is highly suggestive of a pelvirectal abscess.

Diagnosis.—The diagnosis is made on finding digitally a boggy, possibly fluctuating mass through the rectum. This is felt anterolaterally or laterally. A small abscess well above the anus may make digital examination inconclusive. In women, rectovaginal examination is, of course, helpful.

Treatment.—A vertical or curved incision an inch or longer is made about an inch lateral and posterior to the anus. A straight forceps is inserted through this incision and advanced to the levator where a definite resistance is encountered. A finger in the anus guides the forceps and avoids injuring the rectum. The forceps is then passed just through the levator and opened. After evacuation, the cavity is drained by a gauze strip. The drain is removed a day or two later. A finger is inserted subsequently to make sure the opening in the levator is adequate. Hot sitz baths are continued for one and one-half to two weeks.

Retrorectal Abscess.—This is in the retrorectal space, a potential space between the sacrum and the rectum. Rectal infection is the usual cause from an infected crypt or trauma.

Symptoms.—The patient may complain of a dull ache or pain about the sacrum,

may not have been evidenced externally; (2) or an infection that progresses along the lymphatics which accompany a branch of the inferior hemorrhoidal vessel across the ischiorectal fossa. In the latter case, the original infection may be in a lateral, or more rarely, an anterior crypt.

Suppuration in the ischio-rectal fossa may extend posteriorly around the anus at or just above the anal canal to the opposite side forming a horseshoe abscess.

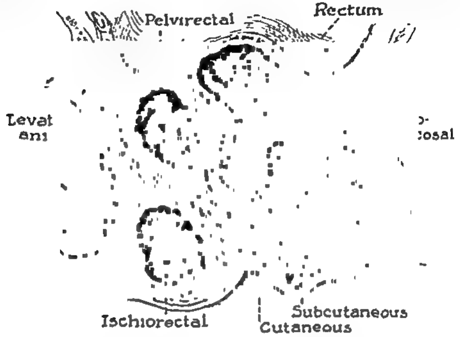

FIG. 675.—Anorectal abscesses.

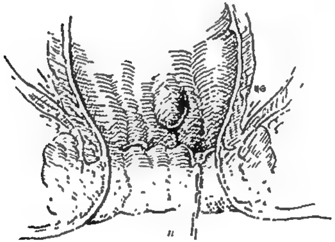

FIG. 676.—Submucous abscess.

It may pass through the levator ani muscle posteriorly and form a retrorectal abscess or pass anterolaterally producing a pelvirectal abscess. Infection in the perianal region may extend by following the superficial part of the external sphincter muscle and its lymphatics, this portion of the muscle lying in a circular gutter of fascia; this gutter is not always present or definite, however.

Treatment.—A vertical linear incision, about an inch long, is made lateral to the sphincter muscle, over the point of fluctuation. If large, a finger is inserted to break up loculi in the fat as in a breast abscess.

usually within an inch of the anus. First search for it in a crypt, *i.e.*, just above the junction of the skin with the mucosa (pectinate line).

Inspection.—Bear in mind the probable site of the primary crypt infection. If the external opening is within an inch or so of the anus and is posterior to the interischial line a line drawn transversely through the tuber ischii and bisecting the anus transversely, the internal orifice is usually in a crypt in the posterior midline. If the external opening is within an inch of the anus and anterior to this line the internal orfice is probably in a crypt directly in line with it. That is, if the external orifice is at a point that may be described as at 4 o'clock, the internal one will probably be at 4 o'clock. This rule has exceptions, but it is helpful.

Palpation.—Finger pressure is made on the skin about the entire perianal circumference. A definite ridge of tissue, the indurated tract, may occasionally be felt running from the external opening to the anus. When the first joint of the finger is inserted into the anus an indurated or depressed spot may be felt at a crypt site: the internal opening of the fistula. This palpation gives information of great value, especially as one becomes more competent. Pressing the anus between the thumb and the intrarectal finger may more definitely outline the induration. As the finger is advanced, it is swept around the rectal wall to determine the presence of indurated tracts above the anus.

Probe Examination.—Flexible silver wire probes, 19 to 21 gauge, are best for exploring the fistula. They are readily made by "beading" with a Bunsen flame the cut ends of silver wire. Do not use steel probes; a false opening is easily made with them.

Injections.—If the internal opening is not found by the silver wire probe passed through the external orifice, the tract may be injected with methylene blue, indigo carmine, mercurochrome, or other dye. If dyes are used, a cotton applicator is previously placed in the anus or an anoscopic examination of the bowel is made to determine the presence of dye in the bowel. Bismuth paste may be used for the same purpose but it may be blocked and fail to identify the internal orifice. Roentgenographic examination after bismuth paste injection is rarely helpful. By means of flexible probes and injections the internal opening should be definitely identified. If none is found after proper search, it probably never existed, or what is less likely, has healed. The case then is a blind external fistula.

Usually the probe is sufficient for diagnosis; it is inserted fairly easily and on manipulation it passes through the internal opening and impinges against the examiner's finger in the rectum. If the tract is too tortuous or has pockets or branches the probe may not suffice. It is sometimes helpful to make a hook in the flexible probe, or use a crypt hook to insert in the internal opening through an anoscope and then run another probe up to it from the external aperture. The course of some tracts can be determined only by incision, as valve-like segments may prevent the passage of liquids and semi-solids, and a tortuous course may defeat probing.

Treatment.—To cure a fistula-in-ano the internal opening must be identified and removed. Most recurrences after fistulectomy result from failure to do this. Other causes of failure are: overlooking lateral tracts, persistence of infection in an old tract due to insufficient skin excision (unroofing), "bridging" during healing or rarely, general diseases, *e.g.*, tuberculosis, diabetes, syphilis.

Operative and Non-operative Methods.—There is no non-operative procedure which can be regarded as a cure for fistula. Zinc chloride (7 or 10 per cent) paste,

a sense of fullness in the rectum and discomfort or pain with bowel movements. Fever or chills are common.

Pressure between the anus and coccyx causes severe pain; a boggy mass is palpated per rectum. A neglected post-rectal abscess may rupture into the peritoneal cavity, or in either ischiorectal space or rectum; it may invade the pelvirectal space.

Treatment.—A curved incision about one and one-half inches long is made from a point just lateral to the midline and extending outward. This is made far enough from the anus to avoid the external sphincter muscle. A pair of forceps is passed up toward the abscess, under guidance of a finger in the rectum and the abscess opened, then the forceps is spread to enlarge the opening. Further steps are the same as those described under pelvirectal abscess.

FISTULA

Etiology.—An anal fistula usually arises from a crypt or anal duct infection. This may follow abrasion of the mucosa of the anal crypt or an area close to it, from a bit of bone, wood, calcareous or other concretion ingested with food; less often probably from a torn anal crypt, laceration of the base of an enlarged anal papilla, a fissure of the anal margin, a small thrombosis in an internal hemorrhoid, or a small sub-mucous hematoma. The anal duct or crypt, or both, become infected and the inflammatory process extends through the rectal wall and an abscess forms. This may rupture spontaneously within the bowel or on the skin, or the abscess be incised through the skin. Thus an infected tract is left: a *complete fistula* with both internal and external openings, a *blind internal fistula* (more accurately a perianal sinus) with only an internal orifice or a *blind external fistula* (sinus) with only an external orifice.* Fistula may, rarely, follow trauma, *e.g.*, a kick or fall on the buttocks or a subcutaneous fistula may develop from an infected thrombotic external hemorrhoid. But the primary infection is in the great majority of cases in a crypt and its duct and the pathological sequence is that stated above. This deserves emphasis and it is of great importance to keep this in mind when looking for the internal opening. Successful results depend upon laying open the whole infected tract or tracts, particularly the internal orifice.

Unusual Types.—A fistula originating in a rectal stricture may have the internal orifice four or more inches above the anus. A fistula may be associated with rectal cancer. A urethroperineal fistula, the result of a periurethral abscess at the site of a stricture in the urethra in men, may open on the skin anywhere close to the anus. However, these are nearly always anterior to and an inch or so from the anus; they are directed here by the superficial layer of the triangular ligament. These types are exceptions and should not confuse one but should serve only to emphasize the necessity of diagnostic thoroughness. An example of a decidedly unusual variety is the following: There was a short (1 inch) fistula having its external orifice in the skin at the anal margin in the posterior midline, with the internal orifice in a posterior midline crypt. There was also another easily palpable tract leading from this crypt around the right half of the rectum and lying on the posterior aspect of the prostate about 1½ inches above the anus, and which terminated in the urethra.

Mistakes are commonly made in looking too high for the internal orifice. It is

* The classification. (1) Internal Fistula (a) incomplete, (b) complete, (2) Combined or Interno-External, (3) External, is descriptive; the above is in more general use.

tuberculous fistula should not be operated upon has been conclusively demonstrated to be a false and obsolete concept.

Perianal abscess in the tuberculous should be incised and drained as soon as it is diagnosed; hemorrhoids and fissures are satisfactorily treated surgically.

Streptomycin, 0.5 gm. daily, if not contraindicated by deficient renal function or other disease is of great value in these tuberculous cases.

HEMORRHOIDECTOMY

Hemorrhoids are varicose veins with accompanying adjacent tissue changes; exceptions and complications are noted below. *Internal hemorrhoids* are covered by mucosa and lie above the pectinate line (dentate border); the *external* are covered by skin and are distal to this line.

Etiology.—Hemorrhoids result from degenerative changes in which chronic inflammation, venous stasis and their effects: thrombosis, venous rupture, ulceration and necrosis, may occur. A localized vascular weakness, often hereditary, is probably the usual predisposing cause. A low grade phlebitis from infection originating in the anal ducts and crypts may well be the most important exciting cause, but in many cases a phlebitis is not microscopically demonstrable. Other factors tending to cause hemorrhoids by interfering with the venous circulation are pregnancy and tumors of the uterus and adnexia; urethral stricture, prostatic hypertrophy and vesical calculus. Obstruction of the portal circulation in hepatic cirrhosis and cardiac decompensation is well known as a cause of hemorrhoids and as a contraindication of hemorrhoidectomy. Chronic constipation or diarrhea are commonly incriminated, how justly is uncertain. Many cases have none of the above factors present or determinably present, thus only an inference can be made. It is often logical to conclude that a local venous predisposition to varicose dilatation existed, as in varicosities of the broad ligament, legs, and elsewhere. In brief, where many causes are stated the real cause is not known.

Varieties: External Hemorrhoids.—These may be thrombotic, varicose or cutaneous (skin tags). The commonest external type for which the patient seeks the physician is the *thrombotic.* This is a pea to cherry sized tender, generally bluish swelling at the anus. It arises suddenly, often when straining with a bowel movement and a sharp stinging pain may be felt. When the patient with hemorrhoids comes complaining of acute pain this is the variety generally found; of course, the patient complaining of "painful piles" may have fissure, abscess, anal carcinoma or other diseases. Piles to the layman merely means anorectal trouble, as piles are the only anal or rectal disease of which most patients know.

Cutaneous hemorrhoids or skin tags or tabs are usually the sequel of an external thrombotic hemorrhoid, the blood clot having been absorbed and a redundant skin fold remaining. They are to be distinguished from the hypertrophied radiating skin folds seen in chronic pruritus ani.

They may also follow a hemorrhoidectomy, especially when the varices were large and insufficient skin was excised. These are an exception to the rule that hemorrhoids are varices, no venous dilatation is present. They may be removed by simple excision under local infiltration anaesthesia if they become inflammed or if they make it difficult to cleanse the anus after defecation.

Varicose hemorrhoids or *marginal varicosities.*—These are commonly seen in pregnancy and require no treatment as they subside after parturition. They are

used either alone or combined with a multiple stage fistulectomy under local anaesthesia is still used in the "ambulant non-operative" treatment, generally by sectarians or cultists. Some types of fistulae are occasionally treated effectively by this means, but the use of sloughing agents is a blind inaccurate procedure and recurrence of the fistula is common. The paste may cause a troublesome acute inflammation.

Fistulectomy.—An accurate knowledge of the anatomical structure and fascial planes of the anal and perianal region is essential to successful result. The infected tract is accurately incised and the internal opening eliminated. If it is large, of the horseshoe type, the infected channels up to the sphincter may be incised and the incision through the sphincter made later. If the sphincter is not incised it is well to tie a heavy black silk suture in the tract around the sphincter muscle to identify it. To provide free drainage slope the edge of the incisions from the skin toward the tract so that a V-shaped gutter remains. The incision through the external sphincter must be at right angles to the direction of the fibers. Incontinence may result from a faulty scar if the incision is not at right angles. Gray gelatinous granulations line the older fistula. As these are wiped away the dense fibrous wall ofthe tract is exposed. This may be scarified with multiple small incisions to increase its blood supply and thus aid healing: this is not necesary in a small fistula. As the gelatinous granulations are wiped off, search the fibrous tissue for openings leading to collateral branches. Probe every suspicious dot and depression to determine whether it is the orifice of a branch. The extremities of the incisions away from the anus may be sutured but it is safer to allow all the incision to heal by granulation. Packing with gauze strips is generally used for the first week or two depending upon the depth and length of incision. The healing wound must be watched to prevent "bridging" by granulations especially about the external sphincter, particularly its upper margin, near the primary, internal orifice of the fistula. If bridging occurs infected channels may thus be left. Wiping the granulations with a cotton-tipped applicator every day or two will ordinarily prevent bridging. Do not pack the wound too freely or too long as this may cause excess scar tissue. Watchful post-operative care is very important in these cases. Mineral oil by mouth may be required for a few days after fistulectomy to produce a daily movement of proper consistency. The patient should have one formed, neither hard nor liquid, bowel movement a day and the wound should be cleaned afterward. A rectal irrigation with hot water after the daily defecation is desirable for two reasons: the cleansing, and the local effect of the heat, temporarily producing a hyperemia and relieving pain.

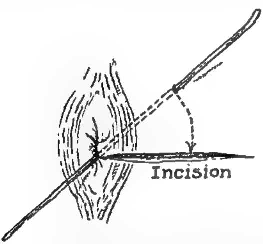

FIG. 678.—Fistulectomy, showing procedure to bring the incision through the sphincter, at a right angle to its fibers. Probe in fistula

Tuberculous Fistulae.—These patients are not operated upon under general anesthesia. They are treated in the same manner as other fistulae. The use of the electrotherm or radio-knife offers no advantage. Because of pulmonary disease and the patient's general condition, the wound may heal more slowly. That

In bleeding or protruding hemorrhoids one formed movement a day is desirable. This may be assured by diet alone or with a retention enema of one to three ounces of mineral, cottonseed, corn (Mazola, Wesson, etc.) or olive oil. Astringents and anaesthetics in ointment may give some temporary relief. Suppositories are an unsatisfactory form of medication as they do not convey medication well down into the folds of the anal canal and are soon melted and the major portion escapes into the ampulla above.

Electrotherapeutic Methods.—Positive galvanism applied with a carbon or leather covered copper electrode is an obsolete procedure.

Electrocoagulation in skilled hands has given satisfactory cures, but a recurrence rate of at least 10 to 20 per cent is inevitable, and in unskilled hands, the recurrence rate is much higher.

Injection methods have the same limitation and the recurrence rate is comparable. In either of the two last mentioned procedures, the recurrences will be much more numerous if the cases are improperly selected. Neither method can be used for external hemorrhoids. Anal skin cannot be safely cooked by an electric needle and the agents commonly used in injections will usually cause pain and necrosis of an external hemorrhoid.

Indications.—Only uncomplicated internal hemorrhoids are amenable to injection therapy. If there is an associated lesion present, *e.g.*, fissure, abscess, fistula, etc., any non-surgical treatment is only palliative. Active or subacute infection in the anus or rectum is at least a temporary contraindication. Marked bleeding is not a contraindication. Striking results may be had in these if the history is short. When prolapse with bowel movements has been present for some time results are less permanent. When prolapse has been common for a year or two, fibrosis in the submucosa about the varix favors recurrence and a permanent result cannot be anticipated.

Technique.—With good exposure of a well-lighted field, a spot on the upper (cephalic) part of the hemorrhoid is swabbed with a cotton tipped applicator, wet with a liquid antiseptic. The needle is inserted at this point into the submucosa. The point should be movable, not into the muscularis where it is fixed, or too superficially into the mucosa. In the latter event the mucosa blanches on injection and necrosis is apt to result. One to two cubic centimeters of a 5 per cent solution of quinine and urea hydrochloride is a satisfactory agent. The same amount of sodium morrhuate may be used. Phenol in oil, cresylic acid and other escharotics are less safe. Three to eight days is the usual interval between treatments. In some cases the period may be shortened. Six to eight injections constitute the usual course. More are sometimes required. A subsequent injection of a hemorrhoid previously treated may be required, not however while infiltration and induration are present.

Permanency of the result depends upon two factors principally; the selection of cases and the number of injections. Less than half of the cases are properly treated by this method, the remainder require hemorrhoidectomy. If the patient is dismissed too soon or fails to return for treatment, recurrences are greater. The criterion of proper hemorrhoid injection therapy is not simply the relief of symptoms, *i.e.*, bleeding and protrusion, but how long such relief lasts. Fifteen per cent of recurrences is not unusual in selected cases; the inexperienced will have a much higher recurrence rate.

Summary.—The injection treatment is indicated only for uncomplicated in-

often evident also in portal venous obstruction, *e.g.*, in hepatic cirrhosis and cardiac decompensation, where hemorrhoidectomy is not indicated. Smith[1] describes these well, "occasionally a patient will complain of protruding hemorrhoids which require digital replacement, but examination will fail to disclose hemorrhoids of sufficient size to cause such symptoms. If the patient assumes a sitting or squatting position and is asked to strain it will be discovered that the protrusion is a swelling of the perianal region caused by subcutaneous varices or external hemorrhoids and instead of replacing a protruding mass, the patient evacuates the blood from the varices by digital pressure. These masses are frequently called marginal varicosities. Treatment consists of surgical excision."

Internal and External Hemorrhoids. Diagnosis.—These are soft, sessile, anal varicose tumors, irregular in shape, size and distribution, each a red to bluish red mass of dilated blood vessels and venous radicals covered by mucosa if internal, by skin if external. The covering being elevated by the varix beneath, the internal hemorrhoid is a definite projection into the lumen of the bowel, the external an equally definite skin elevation. Redundant mucosa seen through an anoscope or proctoscope may be confused with internal hemorrhoids; it is distinguished, however, by being much lighter, the same color as the other rectal mucosa. Neither the history nor digital examination can make an exact diagnosis of internal hemorrhoids. The diagnosis is made by inspection and having the patient strain, often in the squatting posture. Some otherwise excellent surgeons still regularly perform hemorrhoidectomies after digital examinations only in patients with a history of protrusion and of passing bright red blood per rectum. More often than not, they are safe, but an intelligent guess is not a diagnosis and the unhappy results of this carelessness are numerous and should be emphasized. Associated lesions and carcinoma above the hemorrhoids are thus missed; proctoscopic examination is imperative. An accurate diagnosis of internal hemorrhoids can only be made by inspection.

Complications.—Internal hemorrhoids may become infected and superficial erosion or deeper ulceration develop. They commonly prolapse and may be held extruded by the sphincter or in some cases are strangulated with resulting thrombosis and eventually necrosis. Thrombosis of internal hemorrhoids is infrequent unless they prolapse to some degree. External hemorrhoids may be inflammed or a thrombus formed. The clot may be absorbed and usually is if pea sized or smaller, or if larger it may be extruded through an ischemic necrosis of the skin. Occasionally the thrombus becomes infected and a subcutaneous abscess or sinus results. Thrombosis of external hemorrhoids is common.

Treatment.—*Palliative.*—Inflammation or strangulation respond best to hot moist compresses. Judicious use of cold applications gives relief; ice bags are often used inadvisedly. They are apt to cause or hasten necrosis in strangulated hemorrhoids. Some patients obtain greater pain relief from cold applications; if used the patient should be observed carefully for evidence of beginning necrosis.

Regulate the bowels if diarrhea or constipation is present. Diarrhea may cause bleeding from hemorrhoids previously present without symptoms. Relieving the diarrhea may stop the bleeding, at least for some time. In constipation associated with organic disease, a pelvic inflammatory mass or neoplasm, therapeutic measures are, of course, directed to the primary disease. Ordinary constipation demands the proper diet.

[1] Smith, N D.: Text Book of Surgery, Christopher, W. B. Saunders Co., Phila. 1945, page 1093.

is satisfactory; there is less bleeding at operation when a clamp is used. The latter should not be too wide, the broad Kelsey and similar clamps used in clamp and cautery methods may conceal anal lesions.

Clamp and Ligature Excision: Anesthesia—A transacral and caudal block anesthesia is very satisfactory: one per cent novocain, or metycaine solution is used, 10 cc. being injected in each second sacral foramen and 35 cc. in the caudal canal. A low spinal of 50 mg. of either drug dissolved in 1 cc. of spinal fluid and injected in the third lumbar interspace or a liberal perianal infiltration anaesthetic may be used. The sacral block is preferable, the spinal anaesthesia easier to apply. The patient is placed in the Depages' (reverse Trendelenburg) posture. The anus is very gradually dilated digitally, taking three or four minutes. A large Hirschman's anoscope is inserted and held by the assistant. Three or four 3 × 3 gauze sponges, unfolded once to make a 3 × 6 strip are inserted through the anoscope which is then removed and the gauze slowly withdrawn; this aids in demonstrating the size and location of the hemorrhoids. A Van Buren retractor is inserted and the anal canal now well exposed; if there is another lesion, *e.g.*, fissure, hypertrophied papilla, etc., this is taken into account.

An internal hemorrhoid is now picked up by a forceps, the clamp applied at its base, but not including the skin. A suture of No. 0 or 1 plain cat gut is placed through the mucosa at the upper (cephalic) end of the clamp and tied. The assistant holds this out of the way and the tissue on the surface of the clamps is pared off with a scalpel. Two or three sutures are made around the clamp using the original suture. The clamp is loosened and withdrawn and the suture is then tied. An ellipse of mucosa and its subjacent varix has been removed. Next a triangle of redundant skin and its varix, if present, is removed. The apex of this triangle is at the mucosal ellipse, the base being a straight line or curved, depending upon the shape of the external hemorrhoid. A triangular or diamond shaped area of skin is thus excised, usually. Take two or three subcuticular stitches, one on each side of the skin triangle. At times one is placed at the midline of its base, to control bleeding and to bring the mucosal stump down into the anal canal and anchor it there. External pressure will usually control any bleeding which may occur later. It rarely occurs. The same method is followed with the other hemorrhoids. There are generally three or more principal hemorrhoids. Some mucosa and skin must be allowed to remain between the areas of excision. Sometimes a further small hemorrhoid or two may be present. If very small, they may be safely "uncapped": a suture is taken just beyond its upper pole, the hemorrhoid is grasped by a forceps and a little ellipse of mucosa and underlying venous structure is excised; if bleeding continues another suture or two is taken.

Five cc. of an anesthetic agent in oil, *e.g.*, Morgan's solution, xylocaine or other, injected subcutaneously and intramuscularly in the perianal region lessens post-operative pain. It is generally satisfactory but it has not attained the widest acceptance as some surgeons have had unfavorable results.

A three inch strip of folded rubber tissue or Penrose drain is inserted through the anal canal, and a snug dressing of loose gauze over which gauze sponges and a cotton or cellulose pad is placed and a T binder applied. This hemorrhoidectomy is essentially the Earle operation as modified by Buie.

Ligature and Excision Operation.—The internal hemorrhoid is grasped with a straight forceps (Kocher, Rankin, etc.). Another is placed on the skin just distal to the external hemorrhoid. Then a tenaculum grasps the skin just beyond this for

ternal hemorrhoids. At least four weeks of treatment are usually required. It avoids hospitalization and many patients who really need relief and refuse operation will submit to the treatment. The more experienced are more apt to obtain the maximum of permanent results; but even in experienced hands necrosis and mucosal ulcer, usually not serious, may occur. Recurrence after a properly performed hemorrhoidectomy rarely takes place.

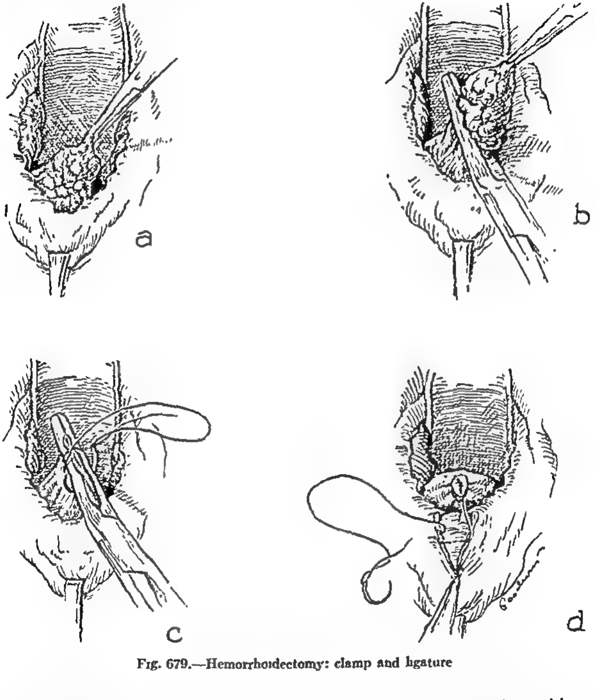

Fig. 679.—Hemorrhoidectomy: clamp and ligature

Curative.—The majority of internal hemorrhoids eventually require excision. Excision either with or without a clamp is current proctologic practice. The obsolete clamp and cautery operation still has advocates, but because of secondary hemorrhage and anal stricture from inadvertent cauterization of the skin of the anal margin, its advocates are decreasing. There are almost as many modifications of the excision and ligature operation as there are of perineorrhaphies, and this applies to the clamp and ligature operation. The last mentioned operation, clamp and ligature

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Ligature and Excision Operation.—The internal hemorrhoid is grasped with a straight forceps (Kocher, Rankin, etc.). Another is placed on the skin just distal to the external hemorrhoid. Then a tenaculum grasps the skin just beyond this for

retraction. A No. 1 plain cat-gut suture is taken with No. 3 Mayo taper-point curved needle through the mucosa at the upper pole of the internal hemorrhoid and tied. One end is left long. The skin and its attached venous, fibrous and fatty tissue is dissected up to the suture and the pedicle thus made is cut. Care is taken to expose and avoid injuring the external sphincter ani muscle; the readily seen transverse fibers are a guide in the dissection. The long end of the suture is used to suture the mucosa to the sphincter. One to three half Lembert sutures are taken through the mucosa and another quite superficially through a few of the fibers of the external sphincter seen in the base of the wound. Another is taken subcuticularly near the apex of the skin excision, 1 or 2 through the mucosa on the opposite side and the stump thus fixed at the level of the upper (inner) margin of the sphincter. Another suture or two may be required for hemostasis. The other hemorrhoids are similarly excised.

ANAL FISSURE

Not every linear break or crack in the anal skin is a fissure. Typical anal fissure considered here (irritable, intolerable ulcer) is practically always single, and in the midline; it is nearly always posterior. It is posterior in perhaps ninety per cent of the cases. Any infection or abrasion of the anal skin may be the cause of a fissure. It starts as a split in the skin of the anal canal and is caused by overstretching in the passage of a large or dry fecal mass. Infection in an anal crypt extending downward and involving the adjacent skin is probably the common remote cause, but the proximate cause is the trauma. A fissure originating as the result of a downward tear of the anal crypt does occur, but a torn crypt is an exceptional cause. The few anterior fissures one sees are generally in women. Some of these are caused during parturition as a result of the child's head stretching the perineum to such an extent that the anal skin tears.

The split in the anal skin may become infected. The lesion commonly becomes a linear ulcer with inflammatory thickening in the edge, and healing is thus delayed. A cross-section under the microscope shows it to be like any other ulcer, with inflammatory debris in the base, small lymphocytes and some leukocytes, infiltrating the edematous edges. The ulcer base is made up of subcutaneous areolar tissue or it may extend down and lie on or in the fibers of the external sphincter muscle.

There are several reasons why a split in the anal skin does not heal readily. Motion and re-infection are important factors. Respiration and abdominal movements are transmitted to the anus and thus it is not immobile more than a few seconds at a time. As immobilization, or at least rest, is important in the healing of any tissue, the anal region is at a disadvantage. Again, the areolar tissue, or sphincter ani muscle fibers which form the base of the fissure come in contact with feces with each bowel movement.

Another reason why some fissures do not heal is the amount of infection in the base; the edges of the ulcer may be undermined and overlie the base so that pus is retained. This approximates a small abscess in the base of the ulcer. Instead of the abscess being directly beneath the break in the tissue of the anal canal, it may lie just to one side of it. This is essentially a small marginal abscess. A small abscess directly beneath the fissure is the more common of the two; both are exceptional. A subcutaneous sinus may extend from the fissure.

Perhaps the greatest importance in the failure of these lesions to heal is the

presence of sphincter spasm. Superficial fissures and some deeper ones heal spontaneously; this is also true of the crack, not a true fissure, which may follow a physician's rough digital examination of the anus, or too marked eversion of the anal skin during inspection. As such cracks are usually out on the skin, not well within the grasp of the sphincter, they are likely to heal readily. Of primary importance in determining whether a particular case under observation may be expected to heal by non-operative measures is the presence of spasm in the anal sphincter. Some surgeons have postulated that an anal fissure which does not heal within three or four weeks under careful treatment should be operated upon. One cannot be quite so definite, but it is a good general rule. Of greater importance than the period of time, however, is the amount of sphincter spasm present. As a result of the inflammatory reaction in the fissure base and edge, the whole sphincter ani is, in acute cases, thrown into a spasm. If the fissure is in the outer portion of the anal canal, it may excite little spasm, hence, healing is not materially delayed. If the sphincter spasm is marked and the fissure is within the grasp of the sphincter, it may not heal even if of short duration. Some fissures of only two weeks duration will not heal because of marked sphincter spasm. Less often fissures older than four weeks may heal in response to local applications.

The presence of a papilla which hangs down into the fissure, a "sentinel pile," a sinus extending from the fissure or a pocket beneath its edges which cannot drain will necessitate excision. The indurated edges of a chronic ulcer must be excised. Treatment can be no better than the diagnosis; the latter must be made accurately before proper treatment can be devised. Digital examination determines the degree of sphincter spasm and may indicate the treatment and prognosis. A satisfactory examination may not be made when the patient is first seen if sphincter spasm is marked and there is much pain, and the means for a complete anal examination are not at hand. A cotton-tipped toothpick applicator saturated with one of the local anaesthetic solutions, *e.g.*, novocain, pontocaine, etc., inserted in the anus and allowed to remain for three or four minutes may permit a satisfactory digital examination. Later, examination under anaesthesia may be required to complete the diagnosis.

TREATMENT

The acute fissure often heals readily under local antiseptic measures with proper regulation of the bowels. Trauma by feces must be prevented. The stool should be neither hard nor liquid. There are probably many superficial fissures which heal spontaneously and which the doctor never sees.

There is often but little sphincter spasm when the fissure first occurs. If the tear is deep, it may occur correspondingly early. Simple measures usually suffice in these cases. Local applications of merthiolate or other antiseptic twice a day and the use of a local anaesthetic ointment, *e.g.*, novocain, pontocaine, etc., may be all that is required. The treatment of the chronic and more severe varieties may be palliative or operative.

Palliative Measures.—Further trauma from the passage of hard feces is prevented by relieving the constipation which is often present. Liquid stools must be avoided. An ointment of 1 per cent novocain, nupercaine or pontocaine applied several times a day partially relieves pain. A chronic fissure can frequently be satisfactorily treated for a time by use of one of the anaesthetics in oil, novocaine, Morgan's solution, etc. These are usually made up as 1 per cent solutions of the

drug in almond or other bland vegetable oil generally with the addition of 5 per cent benzyl alcohol. Five cc. of the solution is injected subcutaneously about the fissure. All fissures can not be treated by this method. More harm than good is usually done with silver nitrate, such cauterization is often too deep or done too frequently. It is most effective if a strong solution (20 per cent) or lunar caustic (stick silver nitrate) is used to coagulate the surface; if the fissure is deep, it is generally of little value.

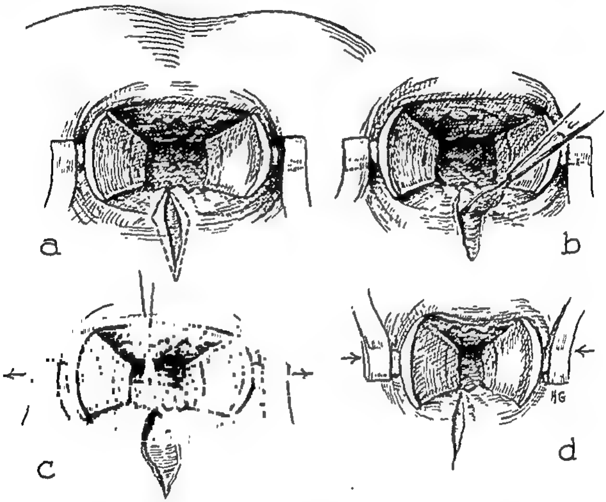

FIG. 650.—Excision of anal fissure.

Digital dilatation occupies an intermediate position. It is not curative in many cases. Whether it is, depends on the existing pathological conditions, such as (1) a sentinel pile, interfering with drainage, (2) an enlarged papilla which falls down and keeps the fissure edges apart, (3) much induration in these edges, or (4) an associated sinus or abscess. In these conditions, simple dilatation should not be expected to cure. Recurrences after ill-advised dilatations are common. Local or gas anaesthesia is used. A dilatation to admit three fingers is done gradually and gently. Anaesthesia is generally necessary.

Operative Measures:—First: Dilate the anus gradually with the fingers, taking three to four minutes to do this. This is not divulsion, which is mentioned only to be condemned One index finger is inserted then the other and they are gradually separated as the sphincter relaxes. Avoid traumatizing the mucosa to a degree which causes bleeding. Dilatation should precede every operation for fissure. Sphincter spasm, which is the usual concomitant of fissure, is thus relieved.

Incision.—A straight cut through the floor of the fissure may suffice. It is inadequate if the pathological conditions described under "Dilatation" are present.

Excision.—This is generally a satisfactory method. It is indicated in the chronic fissure, especially if the edges are indurated or undermined or if there is an abscess associated with it. An ellipse of tissue including the fissure is taken out. If the fissure is wide enough so granulation tissue is seen in its base remove this. The cut should be extended far enough out on the skin to give proper drainage as the wound heals. Failure to do this is a common error. Recurrence is more frequent with a thin narrow scar; thus a sufficient *width of skin* should be excised to give a firm scar. Incising a few of the underlying fibers of the sphincter ani may help to prolong relaxation of the muscle and this aids healing.

If the incision involves the mucosa, one suture will suffice to control bleeding. Do not suture the skin; suturing practically assures recurrence.

Technique.—A suture of No. 0 to 1 plain catgut is placed in the mucosa just above the upper extremity of the fissure.

Traction is made on this suture and an ellipse of mucosa and adjacent skin surrounding the fissure is excised; (Fig. 680). Make sure this is sufficiently wide. Carry the excision well out on the perianal skin; the wound may heal too rapidly at its outer extremity and form a shelf or pocket which drains poorly and delays repair. A gauze strip or wick is left in the wound for twenty-four to forty-eight hours. The usual after-care is given.

CHAPTER 79

PERIPHERAL VASCULAR DISEASES

By W. J. Pickett

GENERAL CONSIDERATIONS

The past several years has seen more and more attention directed toward the clinical phases of vascular disease, especially as it involves the arteries and veins of the extremities.

Many terms indicating the various changes that take place in disease have been employed and many times these have led to a certain amount of confusion.

It is not within the scope of this work to review in detail the etiologic factors and pathologic change which takes place in the common degenerative and inflammatory diseases of the vascular tree, since this has been adequately accomplished in many text books dealing with this aspect of the subject.

Our effort will be directed toward a portrayal of the more common vascular conditions which lend themselves to improvement through surgical means. For the sake of brevity and acknowledging the dangers of over simplification the following classification may serve as a rough guide for further development.

1. Embolic —Embolism
   Thrombosis
2. Occlusive—Thrombo-angitis obliterans
   Arterio sclerosis obliterans
3. Spastic —Traumatic spasm
   Reflex spasm
   Raynaud's disease
   Acro-cyanosis

It is recognized at once that vascular spasm is importantly associated with the one and two groups, and in this regard its control will determine to what extent benefit may be provided through surgical means.

The subject of lymphadema while not commonly thought of as a manifestation of vascular disease cannot be entirely separated from the subject since disease and obstruction of lymph vessels is a frequent associate of thrombo-phlebitis and varicose veins.

EMBOLECTOMY FOR ARTERIAL EMBOLISM

This procedure has been employed by various surgeons since the turn of the century, the earliest attempt being that of Ssabanejew[2] in 1896. Other attempts were made by Lejars[3] Moynihan[4] and Handley[5]. However, it is reported that Labey[6] in 1911 performed the first successful embolectomy. Since that time more and more successful attempts have been reported. The use of anti-coagulants such as heparin and Dicumarol have greatly enhanced these possibilities.

It has been observed that emboli from whatever source tend to lodge at a

bifurcation of the vessel or at a site where the vessel becomes narrowed—*i.e.* axillary to brachial and femoral to popliteal.

Careful palpation of the vessel at these sites plus ossilometric and skin temperature changes will usually reveal the site of the embolism. It is the author's opinion that a preliminary sympathetic block with novocain either at the cervico-thoracic or lumbar regions will relieve the associated vessel spasm and render localization

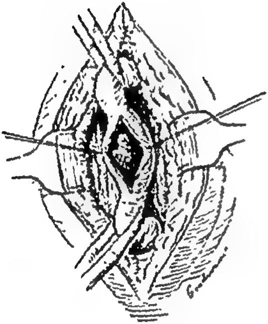

Fig. 681.

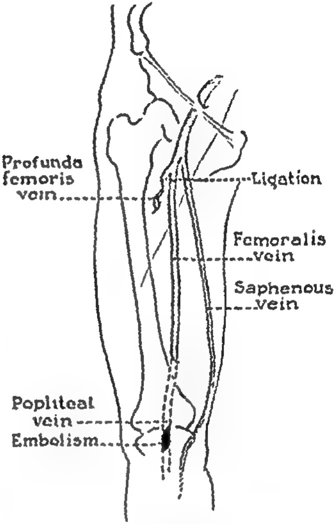

Fig. 682.

Fig. 681.—Embolectomy. Vessel incised longitudinally.

Fig. 682.—Site of ligation of superficial femoral vein to prevent migration of a thrombus.

more accurate. Sudden pain in the extremity associated with blanching of the skin are commonly seen. It must be noted that traumatic spasm of a vessel will render the same picture even though no embolus is present. This latter condition usually responds to vascular sedatives, novocain block, intravenous procaine, etc.

Further evidence of impaired circulation may be determined by the use of the oscillometer, temperature studies, and the radioactive isotopes.[16]

Since the general condition of the patient with embolism is not good, regional or special anesthesia is indicated. Light general anesthesia such as pentothal sodium may be required in some cases.

The vessel is exposed for a distance above and below the site of the embolism. At times the site of the embolism may be determined by a bulge in the vessel wall.

A small soft rubber catheter is passed around the vessel some distance above

and below the site of the embolism. The vessel is opened (Fig. 681) in a longitudiunal fashion. This is superior to a transverse incision since it is easier to close and not prone to tearing.

If the site of the opening is well-chosen, the embolism is readily removed. If there is clotting above and below the site of occlusion these can be removed as a rule by careful aspiration. Undue trauma to the vessel wall should be avoided since this promotes postoperative clotting. The vessel may be irrigated with a solution of heparin if desired and care taken to remove all clots, proximal and distal, since the presence of a small clot left behind promotes further clotting build up.

When a free flow of blood from the proximal and distal segments of the vessel indicates that the occlusion has been overcome, the vessel may be closed with an over an over silk suture attached to an atraumatic needle. The vessel wall should be slightly everted by the suture so that the foreign matter may not impinge upon the interior of the vessel.

At times if the surgeon chooses he may perform a Leriche sympathectomy at the site of the arteriotomy as a means of controlling vascular spasm. Segmental arteriectomy has been employed by some, and might be considered if localized atheroma of the vessel were present as a complication. Heparin is given intravenously in such amounts as are necessary to keep the coagulation time at about fifteen minutes. This is supplemented by Dicumarol the first day by mouth and adjusted to 100 mg. 3× first day and then a daily dose sufficient to keep the prothrombin level within 20 L of normal. If frequent determinations of prothrombin levels are made a daily dose will suffice.

Anti-spasmodic drugs may be employed and daily sympathetic blocks may facilitate the flow of blood through the diseased vessel.

In recent years, the surgeon's efforts have been directed toward the stenosed vessel itself. The abdominal arteriogram has supplied a means of demonstrating occlusion of the iliac and femoral vessels.

Surgery of the vessel itself may consist of endarterectomy or resection of the involved segment, and transplant. Endarterectomy, which encompasses the removal of organized thrombus, thickened intima, and, or atheromatous plaques, through a longitudinal incision in the vessel wall.

This procedure is most effective when the site of the disease is the common iliac vessel. It is most important to know beforehand whether or not the femoral and popliteal vessels are patent, or the results may not be satisfactory.

This can be most readily accomplished by arteriography of the femoral artery as follows: Puncture the femoral artery at the level of the crural arch with a No. 18 needle. Inject 30-50 cc. of 35 to 50 per cent diodrast, in 5 to 6 seconds. Distance 72 inches, exposure 3 to 5 seconds after injection. Inject 5 cc. intravenously 20 minutes before to determine if there is any sensitivity to the drug.

Clinical examination of the extremity, noting color, temperature, absence of pulsations in the peripheral vessels, will inform the surgeon of the extent and progress of the disease.

Claudication, or pain in the calf of the leg on walking 2 to 4 blocks, may mean occlusion of the superficial femoral artery. Pain about the thigh and hip muscles, may indicate occlusion at the common femoral or above. Absence of the femoral pulsation would strongly suggest it. It can be seen that if arteriosclerotic occlusion or calcification has involved the proximal and distal vessels, surgery is not indicated, and may be harmful.

Arterial and venous grafts have been employed to bridge a gap after a segment of vessel has been excised. More recently, various plastic materials have been employed in tubular form as a substitute for vessel grafts. The materials have been for the most part satisfactory, and it has been shown that a cellular membrane, similar to endothelium, will form a lining for these plastic tubes.

If there is tissue necrosis in the extremity, with or without pain, or claudication, then sympathectomy may be contraindicated. Occlusive vascular disease may be treated expectantly. Vasodilators, as Priscoline intravenously, or Roniacol, with a low fat diet, may limit the further formation of atheromas.

Conservative management, with careful avoidance and control of infection, may result in the separation of a necrotic area and retention of a useful limb.

THROMBO-PHLEBITIS AND PHLEBO-THROMBOSIS

In recent years much has appeared in the literature concerning the clotting of blood in the venous system. The use of similar and interchangeable terms has led to some confusion.

Ochsner and DeBakey,[7] Allen,[8] and others have contributed a vast amount of detailed information concerning the mechanism of venous clotting and the resulting danger of pulmonary embolism.

It has been pointed out by Ochsner that the early stage of clotting in a vein is an aseptic process, and that the clot is not attached to the vein wall but rides freely in the venous current.

This stage, phlebo-thrombosis is not of long duration but is by far the more dangerous stage, since walking or any manipulation of the part may result in the detachment of a portion of the clot with a resulting embolism. Post-operative phlebo-thrombosis usually develops in the vein of the calf and may extend along the popliteal and femoral veins into the deep veins of the pelvis.

Venous clotting may develop at a variable number of days following operation. Since its early recognition and prompt management is all important, the surgeon must be continuously on the alert for its presence. Frequent examination of the leg veins are important. Digital pressure on the muscles of the calf and the presence of Homan's sign may reveal tenderness in these regions. Careful observation of the pulse and temperature chart may reveal a slight but sudden rise in either which may be the precurser of this condition. Prompt measures must be taken to control the condition and prevent the detachment of the thrombus and a resulting embolus.

Some authorities favor immediate ligation of one or both femoral veins, while others insist the condition can be managed by use of anti-coagulants. However, most surgeons agree that once pulmonary embolism has occurred, ligation of the veins should be carried out.

Since the popliteal veins are most frequently involved the site of ligation is the femoral triangle. At this site the saphenous vein joins the superficial femoral just above the profunda branch of the vein. The term superficial femoral is not a good one, since this vein drains the popliteal vein and deep veins of the leg. The profunda branch courses posteriorly to drain the upper thigh and muscles of the buttocks. Ligation of the femoral vein should be carried out below the junction of the profunda branch, Figure 682. The vein is transected and a thrombus if present at this level is removed by aspiration. This may be carried out proximally into the iliac vein. It is remarkable how quickly aspirated blood will coagulate in the presence of the

thrombotic clots. Prolonged aspiration should be avoided as it is not difficult to exanguinate a weakened individual to a dangerous degree. If it is not obvious in which leg the thrombus is located, bilateral ligation should be done. If the clotting process has involved the iliac veins, these may be ligated if necessary. Ligation of the inferior vena cava has been carried out successfully, and should be employed if the common iliac veins are also involved. Heparin and Dicumarol are employed in adequate amounts as already described following embolectomy.

Thrombo-phlebitis following injury, infection or surgery is a well-recognized condition. The surgeon frequently sees the condition as the secondary stage of phlebo-thrombosis, which of itself is apparently a short lived condition. The term thrombo-phlebitis implies the existance of inflammation with rise in temperature, pulse and white blood count. This may represent the progression of a localized area of inflammation or it may engraft itself upon a previous sterile process in a not easily explainable manner.

The important feature of this condition is that since the thrombus is more or less firmly attached to the walls of the vein, the danger of detachment and embolus is greatly minimized. The pain and swelling in the extremity can be readily controlled by paravertebral lumbar sympathetic injection of 1 per cent novocain solution as described by Ochsner. Segmental or localized phlebitis of superficial varicosed veins of the sphenous system have sometimes been considered evidence of deep vein involvement. This is commonly not the case, and such phlebitis will not respond to novocain block, since the vein walls are dilated and void of tone. When ambulation is possible an ace bandage should be worn. Lumbar sympathectomy to be described later is often effective in the alleviation of the chronic condition.

SYMPATHECTOMY AND VASCULAR SPASM

For many years surgeons have been interested in the control of vascular spasms, the healing of chronic ulcers, and the control of pain through various operations upon the sympathetic nervous system.

Much of the early writing by Leriche and others,[9,10,11,12,13] had to do with peri-arterial sympathectomy. As was to be expected many of these reports were enthusiastic and the indications for the procedure were greatly broadened. However, as the operation was employed by more and more surgeons the interest and enthusiasm seemed to lag.

In this regard peri-arterial sympathectomy followed the same course that many another operation has experienced in that extensive application by unbiased surgeons has failed to deliver the results announced for it by its original proponents. At the present time the efficacy of this procedure is lightly held by surgeons interested in this field.

The author feels that it has a limited field of usefulness. Its simplicity, ease of application and lack of trauma suggests it as an emergency procedure in the critically ill and the debilitated when some such operation seems indicated. However, if the production of vasodilatation is the purpose of the operation, this can be brought about just as effectively by medical means.

A common criticism of the operation is that its effects, while temporarily appar-

cervical sympathectomy as a means of providing greater effects over a longer period of time.[14]

At the present time lumbar sympathectomy (Fig. 684) is being widely employed not only in the spastic vascular entities but in the occlusive vascular conditions, thromboangitis obliterans, arteriosclerosis obliterans, and chronic lymphedema.

In the former condition, i.e. Buerger's disease, it frequently relieves the pain, possibly by the deliverance of a greater amount of blood to the calf muscles.

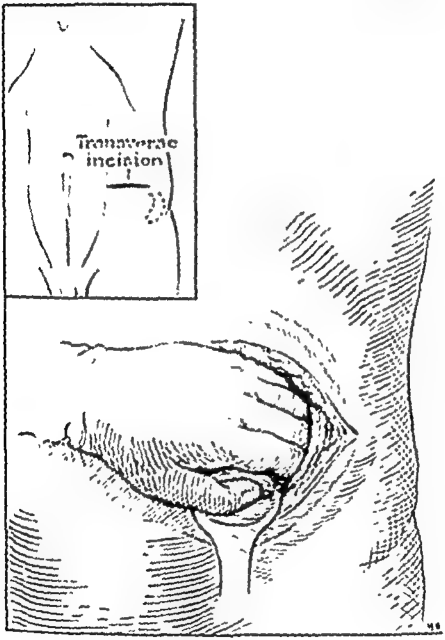

Fig. 683.—Incision for exposure of sympathetic chain.

We have employed the procedure, lumbar sympathectomy, in an effort to forestall gangrene or to limit it in certain cases of vascular insufficiency of both types. In a reasonable percentage of cases it produces the desired results.

It is our practice to carry out a preliminary lumbar sympathetic block as described by Ochsner to determine whether or not the case at hand might be benefitted by sympathectomy. Priscoline intravenously and study of the collateral circulation by means of the radioactive isotope, have afforded us a valuable index of circulatory capacity.[22,33]

There are a number of possible exposures for this operation all of which have their advantages and limitations. The author prefers the transverse extraperitoneal approach as recommended by de Takats. The external oblique and internal oblique are divided transversely along a line from a point 1 inch below and lateral to the umbilicus to a point 1 inch above the anterior superior spine of the ilium. The transversalis fascia and peritoneum are stripped forward with a sponge allowing access to the paravertebral space. This is found along the inferior vena cava on the

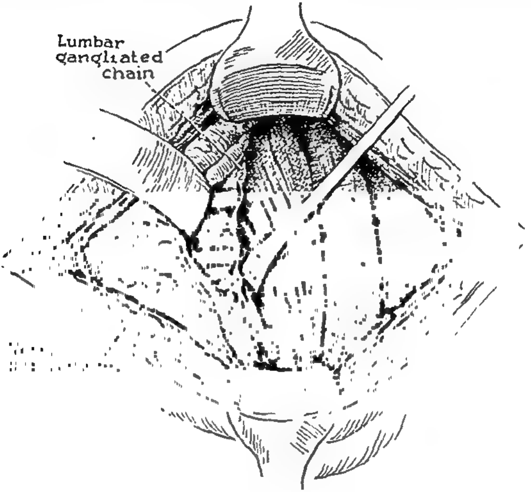

FIG. 684.—Exposure of lumbar sympathetic ganglia.

right side and along the abdominal aorta on the left side. Large Dever retractors are employed to carry the structures away from the posterior abdominal wall. At this point it is frequently easier to palpate the lumbar ganglia with the finger tips than it is to see them. Once located they may be isolated from the surrounding fat and lymph tissue by careful blunt dissection. At times the ganglia may not be separate, but two or more may be fused. The afferent and efferent ramii may be seen and identified. Division of the rami or ramicotomy was at one time employed but we prefer the removal of the ganglia as providing a more effective and lasting result.

Bleeding points are carefully controlled by the use of silver clips, electro-coagulation and often times by simple pressure. The wound is closed in layers without drainage.

CHAPTER 80

VARICOSE VEINS

By Arkell M. Vaughn

HISTORICAL COMMENTS

Varicose veins of the lower extremities is among the most prevalent of the surgical diseases. This disease of the saphenous system, a permanent dilatation of one or more veins, usually with elongation, tortuosity, and thickening of the walls, is specific to man. It is attributable to the upright posture which he assumes. Varicosities have been reported as afflicting man since the beginning of recorded medical literature which reveals that they were recognized and treated since the time of Hippocrates.

Literature on the subject of varicose veins of the lower extremities gives evidence of a vast change and development in methods of treatment. This has been most marked within the past twenty-five years. It has run the gamut of incision, cauterization, multiple puncture, percutaneous ligations, injection, elastic bandaging, and supports such as Unna-like paste boots.

In 1887 Schede introduced the operation of complete excision of the long saphenous vein together with the varicosities. In 1891 Trendelenburg[1] demonstrated Sir Ben Brodie's theory that varicose veins were caused by an incompetent valve in the saphenous vein with a backflow of blood down the vein. He ligated the vein, but the procedure proved unsuccessful. This failure of the Trendelenburg operation was later explained by Schiassi,[2] who showed that the procedure was performed below the fossa ovalis and therefore the ligation was inadequate. Schiassi in 1908 was the first to advise that ligation be combined with injection of the veins with a sclerosing solution. In 1896 Moore[3] recognized the importance of ligating the greater saphenous vein at the saphenofemoral junction. Later, Homans[4] in 1916, and deTakats[5] in 1930 reported success with high ligation, and also recognized the significance of ligating the greater saphenous vein at the saphenofemoral junction.

In 1905 Keller[6] was the first to report a stripping operation. He introduced a twisted wire stripper with a looped end. In 1906 Mayo[7] devised an extraluminal vein stripper. This was a long rigid rod with a ring attached. In 1907 Babcock[8] introduced a stripper with an acorn tip at the end of the shaft. Since that time, many other strippers have been introduced.

In 1908 Friedel[9] excised the greater saphenous vein from the groin to the foot. This operation was later used by many physicians. However, the procedure was gradually discarded because, while the results were good, the postoperative morbidity was prolonged.

Injection of sclerosing solutions alone for obliteration of varicose veins, dates from the invention of the hypodermic syringe in 1853 by Pravaz[10] who attempted to cure aneurysms by injection of ferric chloride solution. Many other European investigators tried various solutions with but little success, until Linser in 1911 discovered that after many injections of mercury bichloride for syphilis, the veins

of the arm became obliterated. Sicard and Gangier[11] observed the same phenomenon in soldiers treated with luargol in World War I.

After these demonstrations, the popularity of sclerotherapy for varicose veins increased, and it was used extensively. This was probably because of the surgical failures and the high mortality rate from pulmonary embolism and other postoperative complications in that early period. Various sclerosing solutions were used for injection of the veins, many of which were later discarded because of their caustic and toxic nature, and sometimes morbidity and fatalities presumed to result from their use.

However, in time, some sclerosing solutions were improved and made safer for use. In 1930 sodium morrhuate was reported upon by Higgins and Kittel[12] and continued to be the most widely used. But serious reactions to its use were gradually reported.[13,14,15,16]

ANATOMY

The venous system of the lower extremities is composed of deep and superficial veins, united by a network of anastomosis. In addition, there are numerous branches and communicating or perforating veins connecting the two. These are variable in number and are equipped with valves. The valves in the saphenous vein, 10 to 20 in number, permit the flow of blood from the superficial into the deep veins, but do not normally permit it to flow from the deep to the superficial veins. These valves, more numerous in the leg than in the thigh, prevent the reflex of blood and maintain the column of blood above them.

The deep veins composed of the femoral, popliteal, anterior and posterior tibial and their tributaries, are located beneath the deep fascia and are surrounded by the powerful muscles of the leg which prevent the veins from dilating when subjected to pressure, while the pumping action of the muscles in walking aids in emptying the veins. For these reasons varicosities of the deep veins rarely occur. The blood from the deep tissues about the foot and ankle collects in the deep veins which become the popliteal as it reaches the popliteal space, and thence continues upward to Hunter's canal where it becomes the femoral, until it passes under Poupart's ligament.

The superficial veins lie in the subcutaneous fat of the leg, forming a network, and consist of the greater and lesser saphenous veins and their tributaries. These veins lack the muscular support and pumping action of the muscles possessed by the deep veins, and therefore, may dilate under excessive pressure to form varicosities.

The greater saphenous vein originates in the medial vein of the dorsum of the foot. It progresses upward superficially on the median surface of the lower leg and thigh in relation to the saphenous nerve, until it empties into the femoral vein about 3 centimeters below the inguinal ligament at the fossa ovalis.

There is usually one large branch just below the knee, superficial to the greater saphenous vein proper. Between the knee and groin the medial femoral cutaneous vein is quite constant and the lateral femoral cutaneous vein less so. Many variations are encountered in the venous pattern in the region of the fossa ovalis. The most commonly encountered veins are the superficial external pudendal, of which there are usually two; the superficial epigastric, superficial iliac circumflex, and occasionally, as previously mentioned, the lateral femoral cutaneous. At the lower

the dilatation. Some patients with small veins may have definite symptoms, while others with markedly dilated veins may have none. The most frequent complaints are heaviness and a tired feeling in the extremities, especially after standing. General fatigue is also a frequent symptom. Sometimes pain resembling a cramp, and burning, itching, tingling, and edema are occasional symptoms.

DIAGNOSTIC METHODS

A careful scrutinizing examination of the involved leg prior to treatment is stressed as a primary pre-requisite to successful surgery or any other form of therapy for varices of the lower extremities. There are various diagnostic methods, and several tests used for determining the patency of the deep veins, valvular incompetence, and disturbed deep venous circulation. The tests most widely used today include the Trendelenburg, the Perthes', the Ochsner and Mahorner, and the Schwartz percussion test. The methods of carrying out these tests, and the manner of interpreting the findings follow:

Perthes' Test.—This test is highly informative regarding the patency of the deep veins. The saphenous trunk is constricted with a tourniquet and vigorous muscular exercise is carried out. If the deep veins are patent or if standing does not increase pressure in them, the exercise will cause the veins to collapse. If the superficial veins do not collapse, this is evidence that the deep veins are not patent or that pressure is greater than normal.

Trendelenburg Test.—With the patient lying supine, the leg is elevated, allowing the blood to flow out of it. A constrictor is applied high on the thigh, or firm pressure is applied to block the saphenous vein. If, with the patient standing, veins in the calf remain collapsed, influx of blood occurs only from above. If they fill quickly it indicates incompetent communicators from the deep to the superficial veins. In other words, if the veins remain empty for 30 seconds or more and refill promptly from above on removal of the constrictor, the results may be assumed to be positive. On the other hand, filling from below with the tourniquet in place represents an inability of communicating veins to control the influx of blood from the deep veins. These results are negative.

Ochsner and Mahorner Test.—This is an exercise test with a tourniquet applied at various levels of the lower extremity, with the patient walking a prescribed course. Prominence and emptying of the veins is observed. If the deep and communicating veins are patent, there is diminished prominence of the veins. When the tourniquet is below the lowest communicating vein and the valves are incompetent, the varicosities at the calf collapse or disappear, and the level and degree of incompetent communicators can be assessed.

Schwartz (Percussion) Test.—With the patient standing, one hand palpates over the saphenous vein at the groin; the other hand percusses over the course of the dilated veins. If an impulse is noted by the "watching hand," incompetent valves and a dilated trunk are suggested. Proof is established by a reversal of the procedure. An impulse felt below indicates incompetent valves.

Opinions differ to some extent as to the comparative merits of the foregoing testing procedures. In the opinion of the present author, the Perthes' test should be done on all patients. In addition, it is of value to obtain a positive reaction to a Trendelenburg test before attempting any corrective procedure for varicosities of the lower extremities.

TREATMENT

Although there is a difference of opinion regarding the choice of methods for treating varicose veins, the trend during recent years has been and is today away from injection therapy, and with an emphasis on operative treatment. Of course, in the treatment of varices of the lower extremities as in that of any other disease, progress has been and probably will continue to be made.

As a specific treatment for varicose veins injection therapy alone is being used less and less, although it still has a place after ligation and stripping. The reason for the discontinuance is undoubtedly because of frequent recurrences of the varicosities and certain untoward complications observed and reported after its extensive use, such as pulmonary embolism, severe anaphylaxis, local chemical thrombophlebitis, and lymphedema caused by necrosis of adjacent lymph vessels.

Injection may be indicated occasionally when age, senility, economic reasons, aversion to operation, very small asymptomatic varicosities which do not warrant operative treatment, or other factors contraindicate surgery. The solutions recommended for these injections are: Varisol (invert sugar, sodium chloride and benzyl carbinol) which is the most effective and is used by the author in the larger veins; De-LS Formula (dextrose, lithium salicylate and benzyl alcohol) which is less effective and is used in smaller veins; Sylnasol (sodium salts of psyllium seed oil with benzyl alcohol) and Formula 61 (potassium guiacol sulfonate and tannic glucoside in a colloidal suspension) which the author has found satisfactory for the small spider veins or "bursts."

The evolution in the treatment of varicose veins of the lower extremities has included five phases: (1) injection of a sclerosing solution into the vein; (2) high saphenous vein ligation alone; (3) high saphenous vein ligation with complete resection of the veins at the fossa ovalis, plus retrograde injection of a sclerosing solution through a rubber urethral catheter into the distal stump of the saphenous vein; (4) high saphenous vein ligation with multiple retrograde saphenous vein ligations and phlebectomy, with the aid of a malleable intraluminal guide[20]; and (5) high saphenous vein ligation with complete ligation of all tributaries, combined with stripping of the saphenous vein from the fossa ovalis to the ankle, and multiple ligations of the communicating and perforating veins. This last method is now used almost exclusively by the author, and he has found the long-term results with the procedure uniformly better than with previous methods.

There is increasing agreement on the efficacy of radical removal of the main trunks of the greater and lesser saphenous veins, with ligation of the greater saphenous vein at the saphenofemoral junction, and stripping of the greater and lesser veins from the fossa ovalis to the internal malleolus. Myers[21] of the Mayo Clinic states that "The stripping operation is the best method as yet devised for the treatment of varicose veins. If properly performed it destroys the venous pattern and collateral veins sufficiently well that recurrences or persistences are uncommon, at least for years."

Sometimes, of course, there are certain instances when ligation alone is indicated. In acute thrombosis in the greater saphenous vein, especially above the knee, which is propagating upward, the saphenous vein is opened. If a thrombus is encountered which extends into the common femoral vein a thrombectomy is performed and the saphenous vein is ligated at the saphenofemoral junction.[22] Extension of a propagating clot into the common femoral vein from the saphenous

vein has occurred in 60 per cent of the author's patients operated upon for this condition.

INDICATIONS FOR AND CONTRAINDICATIONS TO STRIPPING

Some of the more important indications for the stripping operation for varicose veins of the lower extremity are: incompetence of the saphenofemoral and other valves in the saphenous vein, venous stasis, previous or recurrent acute superficial phlebitis, and large varicosities.

Temporary or permanent contraindications to the stripping procedure and/or ligation include: deep venous insufficiency, recent or acute superficial thrombophlebitis, acute or subacute stasis cellulitis of the involved lower extremity, recent extensive sclerotherapy, arterial deficiency of the leg, chronic lymphedema with varicosities, metabolic disturbances, weeping dermatitis or suppurative disease, severe anemia, very small and early asymptomatic varicosities especially in elderly patients, normal but prominent appearing veins, pregnancy, and general debility or poor physical condition with poor prognosis.

OPERATION

In varicose vein surgery the pre- and postoperative care is similar in most respects to that for any other type of operation. Blood and fluid replacement are occasionally necessary after operation, and analgesics are given according to the patient's required needs. There is, however, one exception; that of avoiding administration of depressants to varicose vein patients, in order that the desired early ambulation may be carried out whenever possible.

The veins of the legs are usually marked the day previous to, or the day of, operation, preferably by the operator himself. The main channel of the saphenous vein and all tributaries and perforating veins should be carefully marked with the patient standing. The author uses a pyrogallic acid solution as recommended by Myers and Smith.[23] It consists of "pyrogallic acid, 5 gm.; acetone, 50 cc.; solution of ferric chloride N.F. 40 cc., and ethyl alcohol to make 100 cc. The directions for mixing the solution are as follows: Dissolve pyrogallic acid in alcohol; add acetone and solution of ferric chloride and alcohol to make 100 cc. This solution should be kept in a dark bottle and not exposed to the air." The solution is preferred by the present author because it is not affected by vigorous scrubbing of the leg, and he has observed no irritation or other untoward effects from its use.

General anesthesia with intravenous sodium pentothal is given. Sometimes, however, the inguinal incision and preliminary dissection are begun under local procaine anesthesia and sodium pentothal employed during the actual stripping.

The patient is placed on the operating table in the prone position, and a sand bag placed under his slightly flexed knee. This allows the femoral triangle to be outlined. In the stripping procedure it is important that the greater saphenous vein be ligated flush with the femoral vein and that no branches of the saphenous vein be overlooked. Therefore, it is vital that the femoral vein be visualized at the fossa ovalis. The fossa ovalis, which is an important area revealing all of the branches, is at the apex of the femoral triangle. The leg and abdomen are shaved and scrubbed with soap and water from the umbilicus down to the toes, and painted with Zephiran or some similar preparation. The patient is draped, leaving the lower extremity exposed from the groin to the ankle.

TECHNIC OF HIGH SAPHENOUS VEIN LIGATION AND STRIPPING

An incision is made in the groin, parallel to Poupart's ligament, over the apex of the femoral triangle, down to the deep layer of the superficial fascia (Fig. 686, *A*, Incision *I*). This fascia is cut longitudinally and the greater saphenous vein dissected free. Two clamps are placed upon it and the vein divided (Fig. 686, *B*).

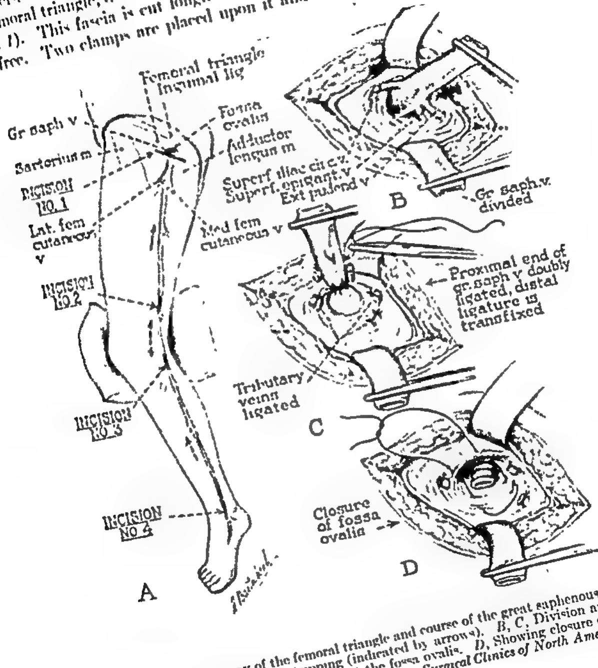

Fig. 686.—*A*, Anatomy of the femoral triangle and course of the great saphenous vein. Location of incision and direction of stripping (indicated by arrows). *B*, *C*, Division and ligation of great saphenous vein and tributary veins at the fossa ovalis. *D*, Showing closure of fossa ovalis with plain 0 catgut (Vaughan, Annan and Caserta in *Surgical Clinics of North America*, Courtesy of W. B. Saunders Company.)

Dissection is then continued downward to the medial femoral cutaneous vein (Fig. 686, *A*). This vein is ligated with catgut, which is the suture material the author is using at the present time. Cotton, however, is also a good suture material for this purpose, if desired.

Next, the proximal end of the greater saphenous vein is followed to the saphenofemoral junction by careful dissection (Fig. 686, *B*). All tributaries are individually

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The veins of the legs are usually marked the day previous to, or the day of, operation, preferably by the operator himself. The main channel of the saphenous vein and all tributaries and perforating veins should be carefully marked with the patient standing. The author uses a pyrogallic acid solution as recommended by Myers and Smith.[23] It consists of "pyrogallic acid, 5 gm.; acetone, 50 cc.; solution of ferric chloride N.F. 40 cc., and ethyl alcohol to make 100 cc. The directions for mixing the solution are as follows: Dissolve pyrogallic acid in alcohol; add acetone and solution of ferric chloride and alcohol to make 100 cc. This solution should be kept in a dark bottle and not exposed to the air." The solution is preferred by the present author because it is not affected by vigorous scrubbing of the leg, and he has observed no irritation or other untoward effects from its use.

General anesthesia with intravenous sodium pentothal is given. Sometimes, however, the inguinal incision and preliminary dissection are begun under local procaine anesthesia and sodium pentothal employed during the actual stripping.

The patient is placed on the operating table in the prone position, and a sand bag placed under his slightly flexed knee. This allows the femoral triangle to be outlined. In the stripping procedure it is important that the greater saphenous vein be ligated flush with the femoral vein and that no branches of the saphenous vein be overlooked. Therefore, it is vital that the femoral vein be visualized at the fossa ovalis. The fossa ovalis, which is an important area revealing all of the branches, is at the apex of the femoral triangle. The leg and abdomen are shaved and scrubbed with soap and water from the umbilicus down to the toes, and painted with Zephiran or some similar preparation. The patient is draped, leaving the lower extremity exposed from the groin to the ankle.

TECHNIC OF HIGH SAPHENOUS VEIN LIGATION AND STRIPPING

An incision is made in the groin, parallel to Poupart's ligament, over the apex of the femoral triangle, down to the deep layer of the superficial fascia (Fig. 686, *A*, Incision *1*). This fascia is cut longitudinally and the greater saphenous vein dissected free. Two clamps are placed upon it and the vein divided (Fig. 686, *B*).

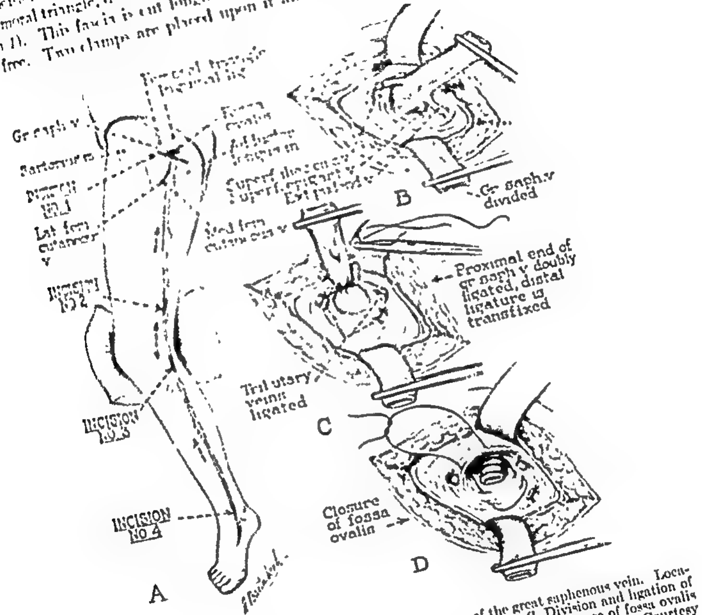

Fig. 686.—*A*, Anatomy of the femoral triangle and course of the great saphenous vein. Location of incision and direction of stripping (indicated by arrows). *B*, *C*, Division and ligation of great saphenous vein and tributary veins at the fossa ovalis. *D*, Showing closure of fossa ovalis with plain 0 catgut. (Vaughan, Annan and Caserta in *Surgical Clinics of North America*, Courtesy of W. B. Saunders Company.)

Dissection is then continued downward to the medial femoral cutaneous vein (Fig. 686, *A*). This vein is ligated with catgut, which is the suture material the author is using at the present time. Cotton, however, is also a good suture material for this purpose, if desired.

Next, the proximal end of the greater saphenous vein is followed to the saphenofemoral junction by careful dissection (Fig. 686, *B*). All tributaries are individually

clamped, cut, and ligated, with plain 00 catgut (Fig. 686, *C*). The saphenous vein is then doubly ligated flush with the femoral vein, with chromic 0 catgut, the distal end transfixed with a suture ligature. The excess vein is excised, the fossa ovalis closed with a purse-string suture (Fig. 686, *D*).

The hemostat on the distal end of the vein is rotated 180 degrees and an incision is now made into the lumen of the vein over the hemostat (Fig. 687, *A-1*). The

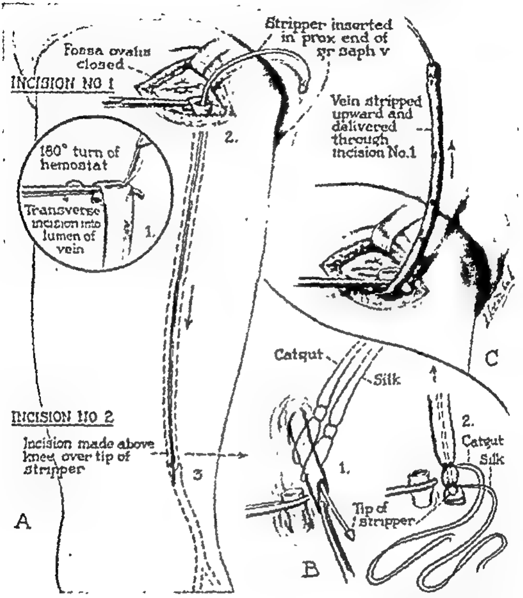

FIG. 687.—*A*, *1*, Transverse incision into lumen of vein over hemostat *A*, *2*, Insertion of ... Showing division ... er. *C*, Vein ... (Vaughan, ... Company.)

stripper is placed into the lumen of the vein, until obstruction is met (Fig. 687, *A*). It may proceed unhampered down the entire length of the vein to the medial aspect of the ankle However, the author prefers making an incision above the knee over the stripper (Fig. 687, *A*, Incision *2*); pick up the greater saphenous vein and dissect, sever, and ligate any communicating veins, and doubly clamp and sever the greater saphenous vein (Fig. 687, *B*, *1* and *2*, Incision *2*). The proximal hemostat is released and the olive tip of the stripper allowed to extend through the

lumen (Fig. 687, *B*). The proximal segment of the vein is doubly ligated on the stripper with a long piece of catgut and secondly with about 18 inches of silk suture (Fig. 687, *B*). These sutures are left long in order that the vein can be traced out to the lower incision, in case the vein breaks while being stripped upward. In such case the vein can be tied over the stripper at its proximal end and then stripped downward. Next the stripper is pulled upward, while pressure is applied over the

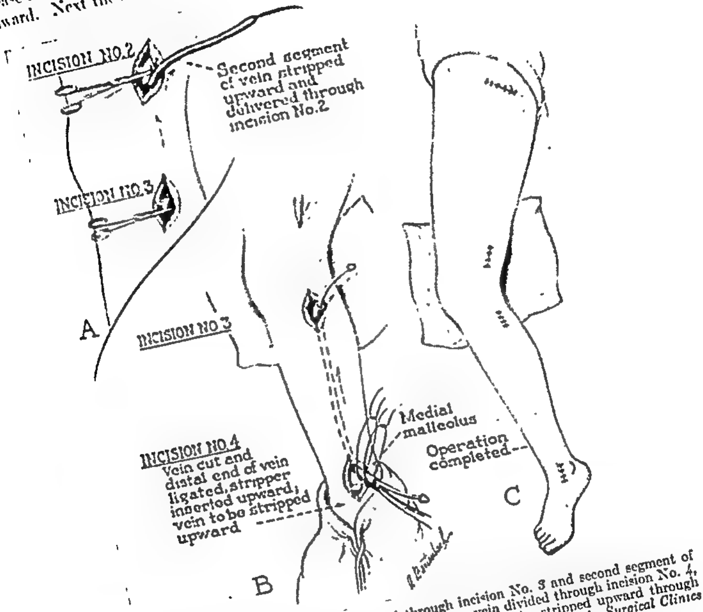

FIG 688.—*A*, Great saphenous vein divided through incision No. 3 and second segment of vein delivered through incision No 2. *B*, Great saphenous vein divided through incision No. 4, distal end ligated and third segment of great saphenous vein to be stripped upward through incision No. 3. *C*, Showing incisions closed. (Vaughan, Annan and Caserta in *Surgical Clinics of North America*, Courtesy of W. B. Saunders Company.)

skin of the medial surface of the thigh. If a large communicating or perforating vein is found, resistance will be encountered, and this vein is ligated through a separate skin incision if large, or pulled off if small. The vein, having been turned inside out, is removed with the stripper from the incision in the groin (Fig. 687, *C*).

Next the stripper is placed in the distal segment of vein and passed to below the knee where another longitudinal incision is made (Fig. 688, *A*, Incisions *2* and *3*).

QUESTIONNAIRE

1. When were varicose veins first recognized and treated?
2. Who introduced the first surgery on varicose veins?
3. Who first advised that ligation of the vein be combined with injection of a sclerosing solution?
4. Who reported the first stripping operation?
5. What was the difference between the Mayo procedure in 1906 and that of Babcock in 1907?
6. Why was the Friedel procedure not a good operation?
7. When was the hypodermic syringe invented and what relation did its discovery have to the treatment of varicose veins?
8. Name some present day sclerosing solutions.
9. What was the most common solution used for injection treatment alone?
10. What complication occasionally arose from injection of sodium morrhuate?
11. Give the anatomy of the greater and lesser saphenous veins.
12. Outline the boundaries of the femoral triangle.
13. Give the origin and course of the greater and lesser saphenous vein.
14. Where is the fossa ovalis?
15. What are some of the theories of the etiology of varicose veins?
16. What is the most recent concept as to the cause of varicose veins?
17. What did experimentation reveal as to the content of varicose blood?
18. Give the symptoms of varicose veins.
19. Describe the diagnostic methods for determining the patency of the deep venous system.
20. Why are these described testing procedures important?
21. Why is the popularity of injection therapy alone, decreasing?
22. When may injection therapy alone still be indicated?
23. Give the phases of evolution in the treatment of varicose veins of the lower extremities.
24. [illegible]
25. Name the most common branches of the saphenous vein at the fossa ovalis.
26. *Under what circumstances is high saphenous vein ligation alone, indicated?*
27. What are the indications for a thrombectomy with ligation?
28. What are the indications and contraindications to high saphenous vein ligation or stripping?
29. What anesthetic is commonly used in the operation for removal of varicose veins?
30. Why are postoperative depressants inadvisable for varicose vein patients?
31. In what position, when, and by whom should veins of the legs be marked for operation for varicosities?
32. What is the author's choice of marking solutions and why?
33. Give the technique of high saphenous vein ligation and stripping as described by the author.
34. Where is the incision near the ankle made for high ligation of the greater and lesser saphenous veins?
35. What nerve must be protected against injury in ligation and stripping of the lesser saphenous vein?
36. Why do varicose veins of the lower extremities occasionally recur?
37. What are the advantages of the two-team operative technique described by the author?
38. When are the skin sutures removed?
39. What postoperative care is given to varicose vein patients?

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CHAPTER 81

PRINCIPLES OF PLASTIC SURGERY

BY WAYNE B. SLAUGHTER

THE term "plastic" as related to surgery has its derivation from the Greek word, "plastikos," meaning, capable of being formed or to mold. It is evident that there is considerable latitude in the interpretation of such a definition and this freedom of application is apparent when the term is considered in light of universal usage.

From institution to institution the meaning of the term varies in detailed description but the following list of uses seems to cover the major portions of the field:

1. Treatment of injuries due to trauma. These are primarily related to the skin and its supportive structures, the bony facial skeleton, appendages, urogenital tract and immediate adjacent tissues.
2. Treatment of congenital defects of head, neck, skin, extremities and urogenital tract.
3. Treatment of tissue losses due to infectious processes or neoplastic changes.
4. Treatment of existing structures or tissues by alteration or movement to enhance appearance, the reconstructive phase of plastic surgery.

In actual practice the demarcation between the various subdivisions is much less concise. To obtain a functional restoration of a part may also involve simultaneous improvement of the appearance of a given individual. In other words, the functional and cosmetic aspect of a given procedure may go hand in hand and are inseparable in assuming the final result in its anticipated maximum manifestation. One of many such relationships may be exemplified in the case of a patient with a cleft lip. Closure of the cleft greatly improves the function of the lip as related to the tongue and teeth, aiding speech and nutrition and eventually aiding in contouring the bony structures of the face resulting in a more normal facial skeleton. At the same time the appearance produces a cosmetic improvement. This concomitant relationship of function to appearance is the rule rather than the exception, as will be pointed out repeatedly in the following text.

To carry out any procedure in plastic surgery it is assumed that the operator is cognizant of the basic embryology, anatomy and physiology which are factors in determining the diagnosis, prognosis and indicated operative manipulation. Success in this field depends upon a thorough knowledge of surgical principles. The unique feature of this specialty is that there are refinements in certain technics that lend themselves to better coaptation of tissue, resulting in minimizing scar, again terminating in better functional and cosmetic habilitation. This, then, is a specialty calling for modification and refinements of certain technics, without deviating from basic fundamental principles as applies to surgery in general.

TRANSPLANTATION OF TISSUE

By the transplantation of tissue is meant the complete or partial separation of a tissue or an organ from its normal connections to a host, and its transfer to a

different location in the same or different individual. Grafts may be classified in many ways depending upon the tissue, its use, its origin and its destination knowing that the more differentiated or specialized a tissue, the less likelihood of its success as a graft.

A classification as to host and donor sites may be useful. The following is one such classification.

1. AUTOGENOUS GRAFTS:

Tissues transferred to different sites in the same individual. The host supplies the graft and is also the recipient (for example, the transplantation of a split skin graft from the abdomen to an ulcerated area on the leg of the same individual). Autogenous grafts may constitute a variety of tissues such as skin, bone, cartilages, cornea, nerve, fascia, tendon, etc.

2. HOMOGENOUS GRAFTS:

Tissues transferred from one individual to another within the same species (for example, the transplantation of cornea from one human to another human). Homogenous grafts are much more limited in application as one goes higher in the phylogenetic scale. In fact, in humans, homografts are never truly assimilated in their new environment, but are merely tolerated tissue in an ideal physiological cultural medium. They are tolerated better if the physiological activity is low and the tissue being transferred is low on the scale in terms of differentiation. At best, homografts can be used with success only when their limitations are understood. The use of cornea and cartilage have limited use in this respect, but, again they may be replaced or actually expulsed by the host. The use of nerve, vessel and bone grafts as a temporary "skeleton" to guide autogenous structures back into their former environment has its indications, but, again they must be recognized in their present state of application as being tolerated foreign bodies, never as functional, physiological structures actually assimilated by the host. One of the useful exceptions, for the use of homogenous tissue is skin. Skin used as split thickness grafts, either from fresh cadavers or viable donors, may be used as a temporary *dressing* to cover *third* degree burns. This may be a lifesaving exercise in extensive burns. Although the "graft dressing" may remain in place from ten to twenty-five days, it also is eventually discarded by the host as an incompatible tissue. But, during the time the dressing is in place, the normal physiological functions of the host may have been restored and the road to recovery continued.

3. HETEROGENOUS GRAFTS:

The transfer of tissue from an organism of one phyla to that of another. Heterografts of any type have, to date, been of insignificant value from a therapeutic standpoint, as far as humans are concerned. Organisms lower in the phylogenetic scale have shown interesting potentialities in respect to heterografts, potentialities that have never been realized in the human. However, certain phases of research indicate some hope for the use of the highly undifferentiated embryonal tissues.

To summarize this phase of the discussion, then, still another useful classification emerges; one that is of value in term of the problem as a whole. This classification is as follows:

a. *Individuality Differential.*—Factors governing grafts that are inherent in each individual, things that make him different from his fellow of the same species. These are multiple and include such things as difference in genes and chromosomal characteristics, metabolites, enzyme products, incompatabilities of various blood components, variation in antibodies and numerous other factors of varying degrees of importance; the sum total of which makes each individual different from his fellow being.

b. *Tissue Differential.*—This term encompasses the differences in characteristics within an individual in relation to the differences of tissue structures and their characteristics as they vary from place to place in normal anatomical distribution. Variation in form and structure of skin, with and without its appendages may be cited as examples.

c. *Organismal Differential.*—This term applies to the organ in relation to its specific requirements and function. For example, the male gonad with its characteristic blood supply, its peculiarities in terms of temperature requirements and finally its secretions, all must meet specific requirements needed of no other tissue in respect to these and other details in order to be transplanted, which status makes it impractical to be transplanted in terms of present-day technique.

In contemplation of a successful graft, these three factors must be satisfied in whole or in part in order to fulfill their anatomical and physiological role in their new environment. It must be further understood that tissues that are transplanted do not undergo metamorphosis in whole or in part to aid in any manner or form, their adaptation in their new environment. If a situation is such that a graft fulfills all the necessary requirements, then the type of graft and the mechanics of the transfer must be considered. These are classified as follows:

1. **Free Grafts.**—These may be of any organ or tissue, but the most successful are of skin, bone, fascia, tendon, nerve and fat although other tissues may be employed. Skin is the most commonly used tissue for grafting, and, with the greatest success. Free skin grafts are: thin split gratfs, intermediate split grafts and full thickness grafts.

In general, the thinner the graft the greater the likelihood of a successful take. Also, the thinner the graft the more it will contract and the more likelihood of it being damaged by trauma since all the protective components of skin have not been transferred.

Thin and intermediate grafts can be removed with a straight edged blade, the skin being held taut by traction, suction or a dermatome. Any device to keep the skin flat so it can be "split" is all that is necessary. Removing skin grafts in this manner gives a reasonably functional piece of skin plus leaving enough tissue at the donor site to supply centers for rapid regeneration of skin and accounts for the practicability of this method.

Full thickness grafts are generally removed by sharp dissection. These full thickness grafts need not necessarily include any appreciable amount of subcutaneous tissue but do include a continouus layer of basal epithelium. The preparation of the skin for grafting consists of thorough washing with a neutral soap and sterile water. The usual aseptic technique is used as in other phases of surgery. No agents that might impair the viability of the graft are used. Protein precipitating agents are particularly to be avoided.

The healing of a full thickness graft in its new bed takes place as follows. In

the first phase, in a twenty-four to forty-eight hour period, there is some fixation by means of plasma gluing. There is no circulation as such but nutrition is maintained by diffusion.

In the second phase blood vessels start to grow into the autograft at about the eighteenth hour. This may be by anastomosis of preëxisting vessels, growth of new vessels into the lumen of old vessels of the graft or by completely new vessels invading the graft. The third stage, that of organic union, takes place about the fifth to tenth day. Changes take place after this, such as contraction and change of color plus changes in physiological characteristics of the transplanted skin. Its relationship to the structures immediately adjacent to it also change as tensile strength increases and the normal processes of regeneration, development and loss

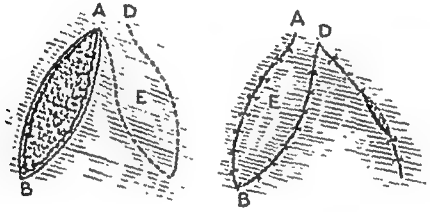


FIG. 690.—Szymanowski's method of simple transposition of a single flap and direct closure of resulting defect. (Davis)

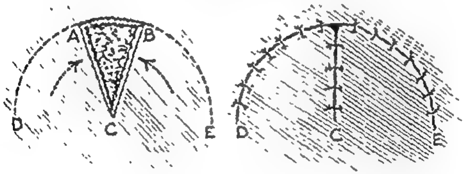


FIG. 691.—Szymanowski's method of advancing a double pedicle to cover a defect (Davis)

of aging cells resume. Color changes are not predictable and generally they either become darker or lighter than the original skin.[1] Generally the lighter the complexion the better the match of the new graft. Contraction of the graft varies in proportion to the thickness of the graft. Axiomatically the thinner the grafts, the more liable they are to contract.

2. **Interpolated Flaps—The Use of Flaps in Grafting.**—A flap is a mass of living tissue that has been dissected in part from its original location but retaining some connection to the underlying or adjacent tissue. Unlike a free graft, it retains a

partial connection with its original site in the form of a pedicle of tissues through which it receives its nutrition until the new blood supply is established at the new site. They are more likely to remain viable, resist infection and have less tendency to undergo contraction and discoloration. They may carry fat and are therefore, particularly useful over surfaces normally exposed to weight bearing or friction such as the heel or the sole of the foot and over exposed tendons, nerves and bones.

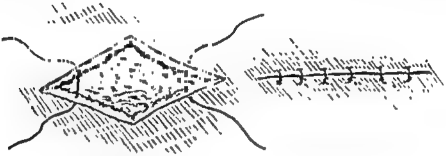
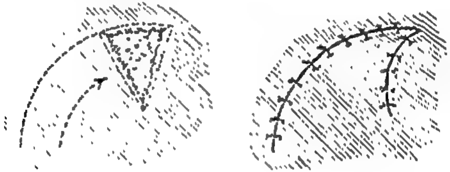


FIG. 692. By undermining adequately various shaped defects can be made into a straight line scar.



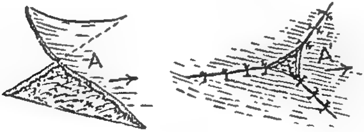


FIG. 694.—V ... a Y-shaped scar ... versed starting ... area.

The main objection to these flaps is that their use may dictate a series of operations and as a rule these procedures extend over a period of time. The flap leaves a defect at the donor site that is not easily corrected and the necessary fixation while the graft is being attached to the recipient area at times is almost intolerable for the patient. Also, the very thickness of the flap may in itself be an objection cosmetically or functionally. For example, when a full thickness graft is placed in the palm of the hand or the eyelid, it may assume the characteristics of the donor site and deposit fat to a degree prohibiting function. Grafted tissues do not undergo

metamorphysis. Circulation in a flap may be increased by delaying procedures wherein the graft is outlined surgically and sutured back in position and elevated in a series of steps. As a rule, it is inadvisable to cross the mid ventral and mid dorsal line in the construction of flaps.

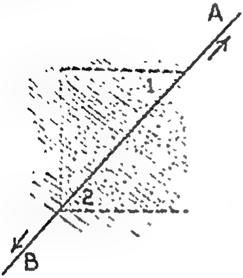


Fig. 695

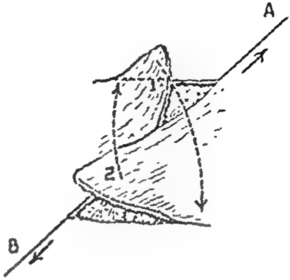
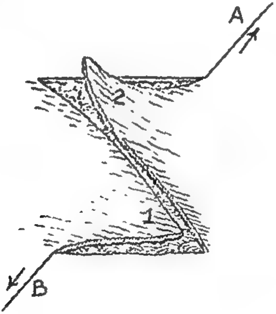


Fig. 696

Fig. 695. The most useful single procedure in plastic surgery is the Z-plastic operation. The line A-B represents the line of tension produced by scar tissue. The object of the operation is to introduce normal, scar-free tissue in this line of tension to impart elasticity to it. The broken lines, placed at an angle to the line A-B, in such a way as to form a Z are the principal components in outlining the desired operation.

Fig. 696.—With line A-B still representing the lines of tension flaps 1 and 2 are made by incisions along the previously determined lines. These flaps must be freely undermined in order to transpose them.



[illegible] tl [illegible] ti [illegible] enough freedom.

Classification of Flaps.—A. *Simple Flap.*—The simple flap is composed of skin with varying amounts of subcutaneous tissue. It retains enough connection to the host to assure viability. The V-Y flap is an example of this as is Szymanowski's simple transposition method (see Fig. 694).

B. *Z-Plastic.*—This last named procedure has as its main attribute, when properly constructed and utilized, the possibility of changing the direction of pull of a scar tissue band in a defect but in so doing does not add nor substitute tissue. These may be single or multiple in character. The Z plastic is the most useful single procedure in plastic surgery when its full possibilities are realized.

C. 1. *Pedicle Flaps.*—These may be an open or tubed graft and they may be single or multiple. They are so constructed that advantage of local blood supply and drainage is incorporated in the graft. They may contain arteries for specific purposes (see Figs. 699–700).

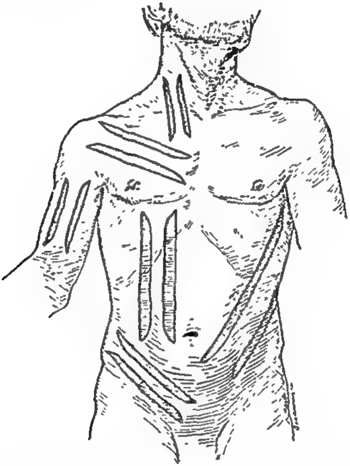


FIG. 698.—Some of the common sites for raising tubed pedicle flaps utilizing the underlying large vessels to insure a more adequate blood supply.

2. *Compound Pedicle Flaps.*—A flap that carries in addition to the skin and subcutaneous tissues, some other structure such as bone or cartilage. Generally used in some form of pedicle flap or tube graft.

3. *Lined or Double Face Pedicle Flap.*—A flap covered with epithelium on both sides and which is employed in cases where a cover and lining are to be supplied simultaneously for repairs of full thickness loss of the lips, nose or cheek. The lining may be made up of tissue of a flap doubled upon itself or it may in itself be grafted with skin or mucous membrane on one side to serve as a lining. The rate of healing and the time of transfer of these grafts depends entirely upon the type of graft, upon the donor site into which it is being placed and its relative blood supply. Stability is maintained in order to prevent motion to allow the growth of new vessels from the host to the donor as in any graft. The accumulation of fluid in dead spaces must be prevented in this type of graft as in a free graft.

FACIAL INJURIES

The adequate management of facial injuries is of paramount importance to the patient from a cosmetic, functional and psychological point of view. The armamentarium of the present day surgeon is such that immediate and proper management of such injuries can greatly reduce undesirable results. The fact that facial injuries are often associated with other bodily trauma that may threaten life itself is often used as an excuse for not treating facial injuries adequately. However, one with a sound clinical judgment can determine to what degree such surgery can be carried out.

Physical Examination. The physical examination of a patient with facial injuries consists first, of an accurate clinical evaluation of the total bodily injury

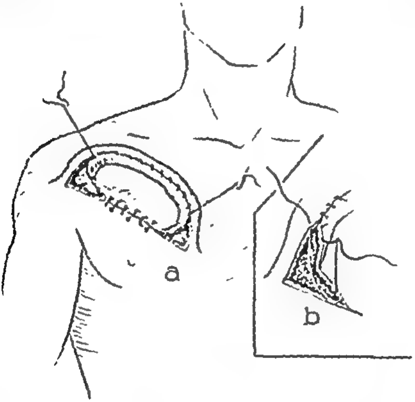


FIG. 671.—H. Gillies method for final closure of the tube (*Amer. J. Surg.*, 1939).

sustained. As these patients often have associated injuries, it is imperative to determine the severity of the injuries in order that those which threaten life itself may be cared for immediately. Sound clinical judgment is just as necessary here as in any other surgical emergency, because much depends on the speed of making a diagnosis so that the necessary treatment may be quickly instituted. Conditions, the control of which may be considered more urgent than the immediate treatment of any facial injuries, are hidden hemorrhage, pain and shock. The judicious use of plasma or whole blood is common in adequate shock therapy. The immobilization of injured parts also takes precedence. No set rules can be laid down because such knowledge comes only with experience, and, varies with each individual and the circumstances attending the injury.

After the general condition of the patient is satisfactory, a systematic examination of the face to determine the extent of the facial injury is in order. The sol

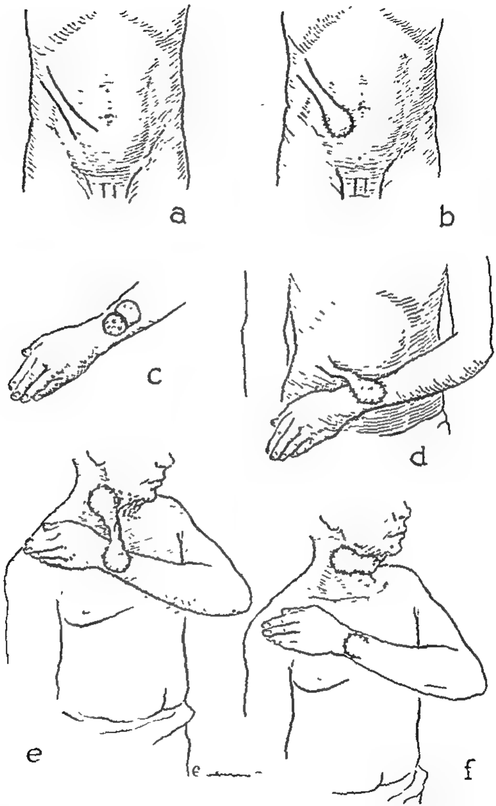


Fig. 700.—Indirect transfer of flaps, when donor and recipient areas cannot be approximated directly. (a) Thoraco-abdominal flap raised; (b) distal attachment cut and resutured back in [illegible]

in its bed (after Gillies).

tissue injuries may be quite evident, but the bony defects may be deceptively camouflaged by soft tissue swelling. In such cases, gentle bimanual palpation is carried out in such a manner that one side is simultaneously compared with the other.

To palpate the bony structures of the face in the best possible manner, one should face the patient in order to compare visually and manually the two sides of the face. The frontal area is first examined. Depression of the frontal bone and especially the bone over the frontal sinuses are hereby compared directly. Then the fingers are brought to bear over the supraorbital rims and moved laterally, eventually palpating the entire orbital rim and always comparing one side with the other. The

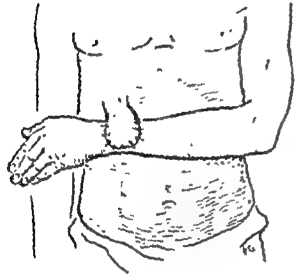


Fig. 701

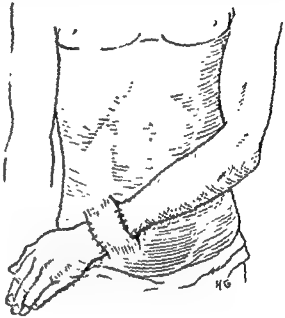


Fig. 702

Fig. 701.—Single pedicle flap from thoracic wall with pedicle above for repair of wrist defect (after Davis).

Fig. 702.—A double pedicle flap from the abdominal wall for repair of forearm defect.

supraorbital notch must not be confused with a fracture site. A displacement of the zygoma will result in a loss of the height of the cheek bone or malar eminence. Next the eyelids are separated and the conjunctiva, cornea and bulbar contents examined. If for any reason the patient is not able to see or the eyeball does not appear normal, the aid of an ophthalmologist should be sought immediately.

The nasal bones are now palpated to determine any loss of symmetry or to elicit motion or crepitus. Any or all of the bones that go to make up the support of the external nose may be involved in a fracture This includes the nasal processes of the maxillae and the frontal bones, and the nasal, lacrimal and ethmoid bones. Fractures of the nose frequently indicate more extensive associated fractures. It is, therefore, necessary to examine the maxilla adequately for possible fractures in every case of nasal fracture. An intranasal examination to determine the state of the bony and cartilaginous septum and the lateral nasal wall is imperative.

The maxillary bone, because of its comparative size, can sustain a great variety of fractures. There is a characteristic distortion of the middle third of the face evidenced by a widening or lengthening, or both. By retracting the lips and examining the alveolar process and the regions above, additional information may be

Parker blade or the point of an aspirating needle to remove these particles individually. When the abrasion is sufficiently cleansed, a fine mesh, mildly antiseptic petrolatum gauze pressure dressing is applied in such a manner as to act as a splint, even though this may require the immobilization of the facial muscles.

Lacerations.—Lacerations about the face are peculiarly different from those elsewhere on the body because the patient desires an acceptable cosmetic as well as a functional result. This fact is further complicated by several specialized structures that must be especially cared for. These structures are the scalp, eyelids, nose, lips and ears.

Scalp lacerations need no particular precaution in their care other than a thorough cleansing with soap and water. It is usually not necessary to shave the hair about the laceration before suturing, but by merely parting the hair in line with the laceration, interrupted sutures can be easily placed, and healing is very prompt. Hair that is shaved may be the source of irritation and a factor in cases of infection as it grows back through the healing wound. If the laceration is of any size, it is advisable that a pressure dressing be applied over the wound for a period of forty-eight hours or longer to prevent the formation of a hematoma under the skin flaps and also acts as a splint during the early healing period.

A type of laceration that occurs rather commonly in the region of the scalp and forehead is the so-called stellate laceration. The force causing this injury is directed perpendicular to the scalp surface and compresses the tissue against the cranial bones. Over the forehead, where the end results of such laceration are somewhat disfiguring to say the least, it is important that a minimal scar be obtained. This type of laceration is difficult to suture with any degree of care because of the multiplicity of the lacerated edges. When it occurs on the forehead, it is best treated by converting the contused, macerated and multilacerated tissue into a single line scar by excision of the fragmented edges and undermining the wound so that the scar will preferably be in the horizontal meridian, following the lines of Langer.

The most important point in the suturing of facial wounds, next to obtaining functioning results, is to obtain desirable cosmetic results. The formation of excessive scar tissue is not compatible with good cosmetic results. In order to minimize the formation of scar tissue it is of utmost importance to remove all possible tension from the skin edges themselves. This is done by adequately undermining the laceration and by placing interrupted buried fine silk, catgut or cotton tension sutures (Fig. 703). If the laceration is from the skin through the mucous membrane surface, this tension suture is a "figure-of-8" suture (Fig. 704) with the knot on the mucous membrane surface. When tissue tension is thus minimized, the skin edges are brought together with a continuous subcuticular stitch of some nonabsorable material such as nylon or non-corrosive metal, preferably on an atraumatic needle (Fig. 705).

Where the skin edges are not in perfect apposition, fine nylon sutures with a friction knot are sufficient to correct this defect. The suture tied in this manner affords some "give" or elasticity to the knot to allow for tissue swelling, and in this manner it tends to reduce scar formation due to localized pressure of the sutures. The normal healing wound has usually reached a state of "serum-glueing," a state of minimum stability, in twenty-four to forty-eight hours, at which time these interrupted sutures may be removed. A firm pressure dressing is then applied over the wound to further aid in splinting the tissues. If the wound is over

a region where bony or soft tissue movement will cause undue tension, the area should be adequately immobilized for five to seven days or longer.

Lid lacerations should be repaired with much care in order to prevent any disfiguring notching, cicatricial extropion, and subsequent exposure keratitis. All types of lacerations are disfiguring but the most deforming scars are most often caused by vertical lacerations involving one or both lid margins. Such lacerations may be caused when a blunt object is thrown against the eye. The anticipation of

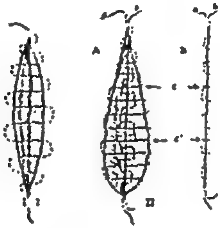


FIG. 705.—Halsted's intradermic or subcuticular suture, employed when especially fine scar is desired. Atraumatic needle intro [illegible] brought out at end. Needle engages corium at [illegible] ersed. Suture then brought out every 2 or 3 cm [illegible] close to skin and at the same level on both sides [illegible] b, superficial suture. B, suture tied, c, c', superficial suture brought out on skin surface every 2 or 3 cm. to facilitate its subsequent removal.

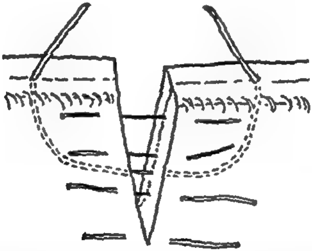


FIG. 706.—Diagram of a V-shaped laceration of lid margin. The first and important stitch is one that is taken in the margin of the lid, beginning 3 or 4 mm. from the lacerated edge on the gray line of one side, to emerge through the gray line on the other lacerated edge about 3 to 4 mm. from its edge. With this suture in place, interrupted 6-0 nylon sutures for the skin surface and fine silk sutures for the conjunctival surface may be readily inserted. A pressure dressing is then applied.

the oncoming blow results in a protective contracture of the orbicularis oculi which thus holds the tarsi rigid. When the blow arrives, the more solid tarsus is split by the force, and the subsequent contracture of the orbicularis oculis pulls the wound apart, thus resulting in the characteristic V-shaped lid laceration. Such lacerations are best repaired by taking a bite with a fine full curved atraumatic needle 3 to 4 mm. from one lacerated edge on the intramarginal sulcus of one side, through the

plane of the tarsus, to emerge through the intramarginal sulcus on the other side about 3 to 4 mm. from its lacerated edge (Fig. 706). With this suture in place but not tied for the moment, it can be used as a traction suture, and the conjunctiva and skin may be separately sutured with greater ease. A petrolatum dressing can then be applied over the lid and a moderate pressure dressing applied. In every laceration about the eye, one should always examine the eyeball and its content to determine the extent of possible ocular injury. If vision is impaired, or if for any reason the eyeball does not appear normal, the aid of an ophthalmologist, should be sought immediately. In the interval, the mere use of a sterile dressing over the eye will suffice as purposeful neglect will do less harm than inquisitive manipulation. Loss of continuity of any portion of the lacrimal duct system may pose a problem in function at a later date. Again it is important to emphasize the fact that there is no time when an opportunity will again present itself when one can do as much for the patient as at the time of the original examination. Resuturing of the lacrimal ducts can be done with varying degrees of success. Inserting a probe into the proximal segment and connecting it with the distal segment is the first step in reconstruction. Suturing the duct with 3 or 4 sutures of 00000 plain catgut on an atraumatic needle is sufficient if done carefully and with the remaining structures sutured back in position in layers over it as added protection. Again, a pressure dressing for stabilization is used.

Lacerations about the nose that involve the cartilages and skin over them require accurate approximation of the cartilage, the skin and the mucosa—all sutured as separate layers. They must be accurately approximated because minor differences in symmetry can readily be noticed. Care in suturing with meticulous approximation of the various layers will usually give satisfactory results. Lacerations involving the tip of the nose must not be heavily sutured as this may impair the blood supply and hinder healing. The presence of deep sebaceous glands in this region invites infection; it is therefore advisable not to bury any sutures here, but simply to close the wound with interrupted or continuous subcutaneous nonabsorbable sutures. Collodion dressing on the surface to further aid in stabilization is indicated.

In vertical lip lacerations one should first approximate the laceration by accurately matching the mucocutaneous line of one side with that of the other. Tension sutures of the "figure-of-8" variety should be placed, and then the skin may be approximated. No dressing is needed over these areas, as any dressing is readily soiled by saliva or nasal secretions. If a dressing is desired, a small collodion impregnated cotton dressing on the skin surface only is satisfactory.

Lacerations of the tongue heal readily and are particularly resistant to infection. Mere puncture wounds, if they are not bleeding, require no suturing. Larger lacerations, especially if they involve the margin of the tongue, usually require suturing. The use of a minimal number of interrupted silk or nylon sutures to adequately approximate the tissues and maintain hemostasis will suffice.

Ear lacerations are difficult to repair because they are usually caused by an avulsive type force so that the lacerated edges are fragmented and beveled. It is not wise to trim any of the fragmented and oftentimes semi-macerated skin, for one will usually be left with an embarrassing insufficiency of skin to cover the cartilage. It is necessary that the cartilage be in accurate apposition before the skin is sutured, otherwise a terraced effect will result from the overlapping. After a careful approximation is made, the next hazard is the prevention of an auricular

hematoma. A dental mold impression of the posterior surface of the ear is made, and a relatively firm dressing may be used over this mold. It is sometimes necessary to weigh the benefits of pressure against the dangers of ischemia, especially when local tissue cyanosis is noted.

It is possible to repair nearly all soft tissue injuries about the face by means of local anesthesia using infiltration or nerve block. The use of local infiltration anesthesia (1 per cent novocaine with adrenalin 1:10,000) does not have any noticeable effect in wound healing or in scar formation.

Fractures.—The management of facial bone fractures may be started at the same time stabilization of the soft tissues is cared for. This may be anywhere up to forty-eight hours after injury, but even after this period one can safely and adequately treat these fractures. The earlier these fractures are reduced, the lower the incidence of infection. This is particularly true of fractures of the mandible.

The severity of facial bone fractures, more particularly the transverse facial fractures, may be such as to entail the combined knowledge of a neurosurgeon, a plastic surgeon and an oral surgeon. In the average civilian practice, however, such cases are rare because when these cases are encountered, the number that survive to receive treatment of their facial fractures is small. In a series of 300 consecutive cases of facial bone fractures treated in a civilian hospital, the incidence of fractures of the various facial bones was as follows:

FRACTURE	PER CENT
Mandibular	86
Zygomatic	5
Transverse Facial	4
Nasal	2
Condylar	2
Frontal	0.5
Coronoid	0.5

A short description of the methods used in the treatment of various facial bone fractures is herewith given. Treatment must be preceded by definite clinical or roentgenographic evidence of a fracture or both, otherwise the situation may be embarrassing. To illustrate this point, a patient with a definite history of trauma to his jaw which was followed by a pronounced swelling and marked trismus, was admitted with a diagnosis of a fractured jaw. The history and clinical finding suggested a fracture at the angle of the mandible, with a deep-seated abscess or hematoma. However, when numerous radiographs were taken, no fracture line could be found. The blood serology was found to be positive, and clinical evidence of a fracture at the angle rapidly disappeared with the proper antiluetic therapy.

Frontal Bone.—A force sufficiently strong to fracture the skull or specifically the frontal bone, may or may not cause damage to the enclosed brain. The most important point to make is the presence or absence of brain damage. If brain damage is present some estimation of the degree and its location is important. The prevention of anoxia by the simple expedient of maintaining an adequate airway may prevent untold permanent damage to the brain substance. The fracture of the brain case is only of significance in that it may signify brain damage and add to its injury by its physical displacement.

A depressed fracture of the frontal bone may be of no particular consequence if the depression involves only the outer cranial plate. If the inner cranial plate is involved and symptoms of cerebral damage are present, the problem is a neurosurgical one. The force that causes a depressed fracture of the frontal orbital rim nearly always makes it a compound one. If the fragment

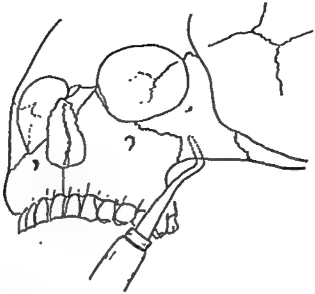


Fig. 707.—[illegible]

Fig. 708.—A representation of a simple fracture through the mental foramen area with an accompanying fracture of the angle of the jaw on the opposite side This illustration shows one of the most universally useful methods of stabilizing jaw fractures. In this case a single strand [illegible] twisted upon itself

downward, it may be replaced by digital manipulation or by open reduction. The exposure of the frontal sinus and also the possibility of the dura being entered, makes the prophylactic use of chemotherapeutic and antibiotic agents imperative. The surgical replacement of a depressed fragment is usually all that is necessary when there is little or no muscle pull to displace or accentuate the defect. The use of an appliance applied externally is only rarely indicated. If a depressed fracture of the outer plate of the frontal bone is seen several days after displacement, and there are no evident complications, it is safe to permit the wound to heal; and at a

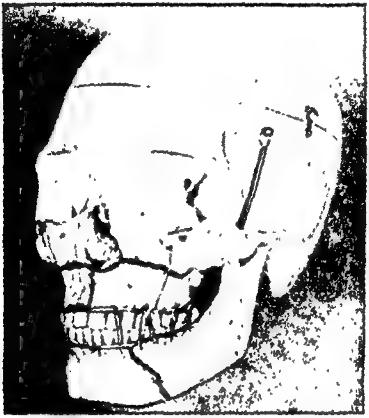


FIG. 709.—This technic, popularized by Adams, shows a method of satisfactory reduction of fractures involving not only the lower jaw but also a transverse fracture of the maxilla. This method combines the method described under Figure 708, in that the upper and lower jaws are stabilized by intermaxillary elastic traction, but, in this case a half round arch bar is contoured to the dentition of both the upper and lower jaws. This is then again stabilized by using single strands of [illegible]

[illegible] ...ances

later period the frontal defect may be filled with autografts of medullary bone from the crest of the ilium or grafts of diced cartilage, contoured to shape, or by utilizing rib bone suitably contoured.

Zygomatic Bone.—A depressed fracture of the zygomatic bone may cause marked facial asymmetry which results in the loss of the height of the "cheek bone" and the distortion of the orbital contour. The force that causes this type of injury does not usually create a compound fracture. These fractures may be safely reduced even several days after being displaced. In the average case reduction is effected by placing an elevating instrument beneath the bone, and then raising the

fragment into position. This instrument may be inserted either through a stab incision in the buccal fold next to the third molar and thence under the malar bone (Fig. 707), or by Gillies' method, through a small incision in the parietal region in the hairbearing area. The instrument is made to slide over the temporal muscle and then under the zygoma. In the latter maneuver, the use of a suitable object under the elevating instrument as a fulcrum will greatly aid the manipulation. In recalcitrant cases it may be necessary to do an open reduction and anchor the zygoma to the frontal bone with wire.[2]

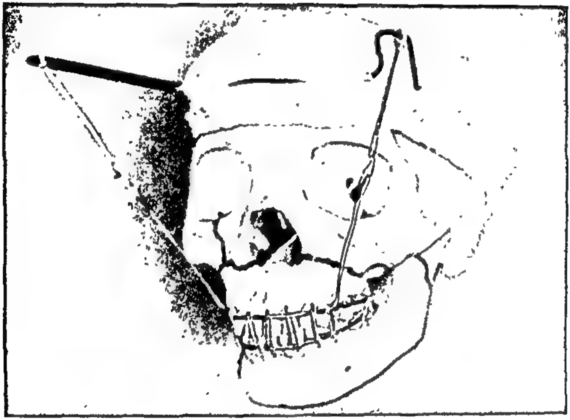


FIG. 710.—The same basic problem presents as shown in Figure 708. However, due to delay [illegible]

Nasal Bone.—A depression and lateral displacement of the bridge of the nose is the usual type of nasal fracture. Such cases are readily corrected if seen early. For anesthesia, the nasal cavity is packed with a 4 per cent cocaine pack, and this is supplemented by a subcutaneous injection of 1 per cent novocaine over the bridge and lateral walls of the nose. The cocaine packs are then removed, and a rubber-protected hemostat or similar instrument is inserted into the nasal cavity and the depressed fragment elevated and maneuvered into position by digital manipulation. An intranasal splint is placed under the depressed fragment, and an external molded splint is placed to hold the fragments in place. The intranasal splint or packing is removed between forty-eight and seventy-two hours later, to prevent pressure

necrosis of the nasal mucous membrane. The external splint may be left on for a week.

In severely compounded and depressed nasal fractures, where it is not possible to use any externally molded splint, it may be necessary to use a U-shaped appliance with one of the arms of the "U" holding up the depressed fragment, and the other arm attached to a headcap appliance. If such an appliance is not immediately available, it may be a wiser procedure to reduce the fracture in as simple a manner as possible and suture the skin over it with interrupted sutures. After the wound is healed, a rhinoplastic procedure may be performed.

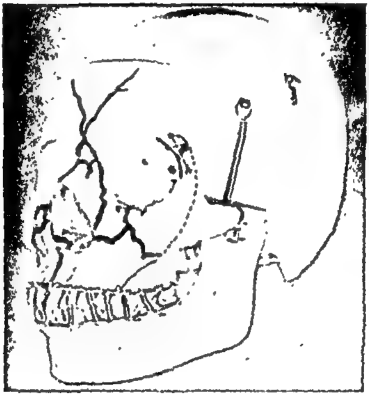


FIG. 711.—Again demonstrating the use of the teeth as a basis for the stabilization. In this [illegible]

Maxillary Bone.—Fractures of the maxilla may vary from a simple puncture wound of the maxillary sinus to a severe impaction or gross displacement. Such fractures may tax the mechanical ingenuity of the surgeon to construct a proper appliance for fixation. The problem is further complicated in an edentulous patient. No single method can be used for all types of fractures, but each case must be studied in its own light. If mechanics of the fracture is understood, the treatment will be evident. The multiplicity of methods of treatment is evidence of this fact.

In a simple puncture wound of the maxillary sinus, the wound is cleaned and freed fragments are removed. The skin over the area is then carefully sutured. No gauze should be packed into the sinus cavity as this would interfere with the normal action of the ciliated epithelium. If there is a pronounced comminution, gauze

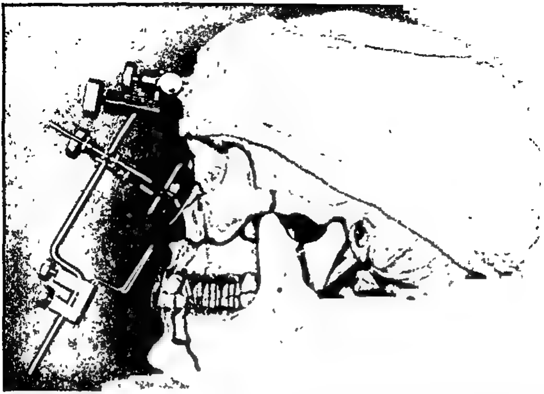


Fig. 712.—In cases where the fractures are seen late and it is difficult to reposition the bony fragments, some type of external traction is necessary. In this case, as in Figure 710, a headcap was applied to hold an appliance by means of which accurate positioning of the nose could be mainta
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rim. T
tendenc
is rarely indicated.

Fig 713—A possible skull fracture is no excuse for delay in treating facial injuries. This

packing may be used to hold the fragments in position. The packing may be removed gradually over a period of days.

In a severely impacted fracture of the maxilla, one can sometimes move the fragment by simply grasping the teeth and loosening the impaction. If this is not possible, one must resort to the use of traction with rubber bands over properly constructed arch bars using the teeth for fixation. When reduction is obtained, fixation is maintained by wiring the teeth of the maxilla to those of the unfractured mandible, the occlusion of the latter being a guide to the proper reduction.

Fig 716

Fig. 717

Fig 716 —Tramatic "saddle nose." Failure to reposition fractured nasal bones can result in this type of a deformity.

use of cartilage ver, this lengthy

In marked downward displacement of the maxilla, a device to fit the entire upper arch is inserted and arms attached in such a manner as to extend out of the mouth and back along the cheek. These arms may then be stabilized by attachment to a head cap, or a half round arch bar is wired to the buccal surface of the maxillary teeth. A loop of stainless steel wire is then placed around the arch bar in the region of the second premolar tooth on each side. The wire is carried upwards through the buccal fold and made to emerge just lateral and inferior to the outer canthus. Local infiltration of novocaine makes this procedure relatively painless. The free ends of the wires are then attached to a bar in the plaster headcap, and the traction is made in the direction that will best correct the displacement, by twisting the ends of the wire over attachments in the headcap. Once occlusion is obtained, it may be maintained by proper wiring of the teeth with the use of the mandibular teeth as a guide and splint (Figs. 713, 714, 715).

is a broad band, it may be excised and made into a linear one or replaced by a full thickness free graft or a pedicle graft.

The use of autografts of cartilage or medullary bone in the reconstruction of bony contour of the nose, chin, forehead and cheek bone is becoming increasingly popular. Cartilage homografts have indicated usage under limited circumstances.

FIG 718 —A, The alliance of functional rehabilitation and cosmetic acceptance is illustrated in this case A marked prognathism of the lower jaw prevented satisfactory occlusion of the lower and upper teeth Measures demonstrated a normal upper jaw with a prominent nose *B*, The … This is done after proper re- … the horizontal … eeth. *C*, Final … he entire facial … nction of both the dentition and nasal passages is improved

The problem of the proper management of facial injuries in all its aspects covers a broad expanse of surgical principles. In cases involving trauma emphasis must be placed on the axiom that there is only one chance to be of maximum help to

the patient, and that is the first time he is seen. So much depends upon what is done at this time, that it will determine to large extent the post-traumatic morbidity. Early and proper treatment should be within the patient's reach even if several personalities must contribute to this end. In cases of congenital defects (Fig. 719) in which reconstruction problems are based not only upon the immediate problem but on those involving the growth potential of the individual, the consideration of injury to growth centers must be considered.

Fig. 719. [illegible] There is marked protrusion and rotation of [illegible]
ba[illegible]
of [illegible]

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